SECONDARY DEGENERATIONS

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SPINAL CORD,

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SECONDARY DEGENERATIONS OF THE SPINAL CORD.

Comparatively recent observations in nervous diseases have proved that when a nerve fibre is injured in any part of its course, it undergoes certain degenerative changes in that portion which is cut off from its connection with the nervous centre, from which it derives its supply of nerve force. In other words the alteration commences at the seat of the lesion and proceeds in the direction of the transmission of nervous force. If, therefore, we have any disease interesting the brain or upper part of the spinal axis, by which certain fasciculi of nerve tubes are destroyed, we can trace these degenerated tubes throughout their course along the spinal cord, observing with accuracy whether they terminate in the gray matter of the cord or pass off in the anterior roots of the rachidian nerves. In the same manner lesions of the posterior roots, or lower segments of the cord result in such alterations of the posterior columns as to admit of our tracing the injured tubes in a centripetal direction.

It being well proved that the ultimate nerve fibres do not anastomose with one another, but continue their course distinct and separate from their peripheral to their central extremity, we have a reliable method of studying each individual nerve throughout its passage along the cord, as well as of investigating its relations to the central ganglia, by carefully observing the alterations which are secondary to primary lesions of the nerve tissue.

This power of following a nerve tube along its course

is of inestimable value in investigating into the pathology and physiology of the nervous system; and accurate observations of cases will doubtless result in a great advance in our knowledge of this complex and difficult subject.

I have translated the following work of Bouchard upon secondary degenerations of the cord, in hope that it will lead those who have opportunities of meeting with such cases to keep careful records of the symptoms and post mortem appearances, which, when tabulated and compared with one another will doubtless serve to elucidate some of the difficult problems which are so continually presented to us by the nervous system.

ALBANY, Nov. 22d, 1868. E. R. HUN.

Besides the lesions proper of the spinal cord which result from a primitive alteration of its tissue, there are others which occupy, as a rule, the whole length of the rachidian axis, which develop very rapidly and almost at the same time throughout this whole extent, and follow either primitive lesions limited to one point of the cord itself, or lesions of the brain, or lastly alterations of the posterior roots.

These secondary degenerations of the cord take no part in the proceedings of the primary lesion. They have their own physiology and pathological anatomy, their special course, the same in every case, whatever be the nature of the diseases of which they are the consequence. They result from that property common to all nerve tubes which, injured at one point of their course, become altered throughout all that portion which has thus lost its relations to the parts from which they take their origin, and which exert a preponderant influence on their nutrition.

Thus interpreted, these alterations of the cord well deserve the name of *secondary degenerations*, and differ essentially from other affections also described as secondary, but which are only the radiations to the cord of a process developed in a neighboring part, such as myelitis or sclerosis consecutive to spinal meningitis; such also as those diffused scleroses of the cord so frequent in general paralysis,* and of which M. Magnan has demonstrated the relations with the cerebral lesions peculiar to that disease.

I should say, at the commencement, that these secondary degenerations of the cord very often escape even a careful examination. Notwithstanding when the attention is drawn to this point we not infrequently discover them with the naked eye. In all cases, certain anatomical processes which I shall explain hereafter, always enable us to recognize them easily. This research, I think, should not be any longer neglected, for the secondary degenerations have their own symptoms independent of those of the primary disease; they may persist after the disappearance of that disease, and then betray themselves by permanent functional disorders which, if one was not forewarned, might be attributed to another cause; lastly, they tend to elucidate certain points still very obscure in the physiology, and above all, in the normal anatomy of the spinal cord.

Was it not for the difficulty of proving them in the absence of the means which we employ at this day, one would have the right to feel astonished that the secondary degenerations of the spinal cord had not struck the observers who have studied the lesions of this nervous centre before the present time. Their discovery is in fact of recent date. In going over the ancient works

* Etudes cliniques et anatomo-pathologiques sur la paralysie générale; prix de l'Académie de Médicine, 1865.

on this subject, I have found but one fact which is connected with it: it is given in the Sepulcretum.* It is in reference to an atrophy of the left half of the cord in a case of a considerable lesion of the right hemisphere. But the author of this observation, to which I shall refer again, Wepfer, knew not how to interpret it. Not only he did not know that a hemiplegia of the left side can be due to a lesion of the right hemisphere, but still more, did not attribute any importance to the lesion of the cord because the arm alone was paralyzed.† Morgagui,‡ who has commented at length upon the observation of Wepfer from a point of view different from that which we now occupy, appears to have noticed the relation which exists between the lesion of the brain and that of the cord. He says, indeed, in the few lines which he devoted to this last alteration, that the lesions of the right hemisphere, "*diminuérent aussi pendant longtemps l'afflux des esprits dans la partie gauche de la moelle épinière.*"‖

It is in fact to Mr. Cruveilhier that the honor of having discovered the descending secondary alterations consecutive to cerebral lesions, is due. He has followed them in the peduncles, in the pons and in the bulb, but he has not recognized them in the cord. I wish to cite this passage according to the text.§ "The attention of observers cannot be drawn too much to the ap-

*Theophili Boneti Sepulcretum, lib. 1, sec. 15, obs. 4, p. 360: Lugduni, 1700.

† Ibid., Scholies de l'observation.

‡ Recherches anatomiques sur le siège et les causes des maladies, traduct. de Desormeaux et Destouet, t. 11, lettre xi., No. 10, p. 116.

‖ Thus diminished for a long time the afflux of the spirits into the left half of the spinal cord.

§ Cruveilhier Anatomie pathologique, liv. xxxii., p. 15.

preciation of the influence of hemorrhage and other lesions of the brain on the condition of the cord, and reciprocally of the influence of lesions of the cord on the condition of the brain. I can give this as a positive fact that the lesions of the cord do not exert any influence upon the brain, but that the lesions of the brain have a very marked action on the cord, both with reference to its functions and its organization. Thus, often coincident with apoplectic cicatrices, a result of the almost complete destruction of the optic thalamus, I have found the anterior pyramid of the same side, and consequently of the side opposite to the hemiplegia, atrophied. This atrophy continued in the prolongation of the pyramid across the pons, and even in front of the pons in the inferior layer of the anterior peduncle. I have not followed this pyramid downward below the decussation. Finally, I have never found, even in the oldest hemiplegias, the corresponding half of the cord atrophied, or at least the difference between the right and left halves of the cord has not struck me, by which I do not mean to say that, a difference which escaped me may not become appreciable to the eyes of a more attentive observer, whose ideas are directed especially to this point."

This observation has given to L. Türcke* the results which M. Cruveilhier had foreseen. In a first memoir, presented in 1851 to the Academy of Sciences of Vienna, he showed the alterations of the cord consecutive to different cerebral lesions, and also to certain partial destructions of the tissue of the cord itself; and he drew from these facts rigorous deductions concerning the

* Ueber secundäre Erkrankung einzelner Rückenmarksstränge und ihrer Fortsetzungen zum Gehirne, dans Compte rendu de la section de mathématiques et sciences naturelles de l'académie des sciences de Vienne, Mars, 1851.

structure and distribution of medullary fibres as well as some physiological consequences much more contestible. Two years later, in a new communication,* he analysed thirteen cases of secondary degeneration in consequence of cerebral lesions, and twelve others resulting from primitive alterations of the cord. Finally, in 1855, he incidentally came upon this question in another memoir.† Notwithstanding their importance, the works of Türcke have not attracted great attention. We find them very summarily mentioned in the treatise of Rokitansky ;‡ they are not even spoken of in that of M. Lebert. At the same time that Türcke published in Germany the result of his researches, analogous discoveries were made in France and in Holland.‖ MM. Charcot and Turner§ presented to the society of biology an example of decussating atrophy of the cerebrum and cerebellum, and noted also a descending atrophy which followed the cerebral peduncle, the pons, the anterior pyramid of the atrophied cerebral hemisphere and the antero-lateral column of the cord on the opposite side. Some years later M. Turner¶ reproduced this fact in his thesis, re-

* L. Türcke, Compt. rend. de. l'Acad. des Sciences de Vienne, t. xi., p. 93, juin, 1853.

† L. Türcke, Beobachtungen über das Leitungsvermögen des menschlichen Rückenmarckes. Ibid., t. xvi., p. 329, Mai, 1855.

‡ Lehrbuch de Pathologischen Anatomie, t. 11, p. 485, 3e édit.

‖ Schroeder Van de Kolk, Waarneming van ene atrophie van het linker halfrond der hersenen met gelïjktijdige atrophie du regterzijde van het ligehaam. Verh. der Eerstê kl. van het Nederl. Institunt, 1852 ; Derde Reeks., D. V., p. 31.

§ Exemple d'atrophie cérébrale avec atrophie et déformation dans une moitié du corps (Compt. rend. de la Société de biologie, 1852, p. 19).

¶ De l'atrophie partielle ou unilatérale du cervelet, de la moelle allongée et de la moelle épinière consécutive aux destructions, avec atropie d'un des hémisphères du cerveau. (Thèses de Paris, 1856.)

porting with it two analogous ones. Similar facts had already been given by Rokitansky.

It would seem that these facts should have had a place in the discussion relative to the alterations which divided nerves may undergo. These two questions, so to speak, contemporary, and which could have thrown light upon one another, were nevertheless developed almost independently. At the same time that Türcke communicated the result of his first researches, at Vienna, Waller* published at Bonn, at London, and at Paris, his experiments on the degeneration of divided nerves. In point of fact his observations did not bear upon the elements of the nervous centres; but the laws of the degeneration of nerve fibres such as he has formulated them seem to me rigorously applicable to our subject. It was also at this time when MM. Philippeaux and Vulpian† made known their researches on the regeneration of nerves, that the study of the secondary degenerations of the nervous centres was undertaken in France. In the case published by M. Gubler,‡ an alteration of this nature commencing at the primary lesion of the brain descended

*We cannot say that Waller discovered an alteration of the posterior columns of the cord as a result of the lesion of the posterior root. He has certainly observed that, when these roots are divided, they degenerate between the point of section and the cord, and even that the alteration advances for a short distance into the substance of the posterior columns; but what he saw here was only an alteration of the intermedullary portion of the roots. Augustus Waller, Nouvelle méthode anatomique pour l'investigation du systeme nerveux (lettre à l'academie des sciences du 23 Novembre, 1851:) Bonn, 1852.

†Sur la Régénération des nerf séparés des centres nerveux. (Memoires de la société de biologie, 1859, p. 343.)

‡Du Ramollissement cérébral atrophique envisagé comme lesion consécutive à d'autres affections encéphaliques (Arch. gen. de med., t. 11, p. 31, Année, 1859.

across the peduncle to the pons. It was not followed further, and the condition of the cord is not noted; but already at this time an observation, cited by M. Gubler, had been presented to the society of biology by M. Charcot. In this case the descending lesion was not only manifest in the peduncle, the pons, and the anterior pyramid of the same side, but it continued in the antero-lateral column of the opposite side of the cord.

Since this time, a large number of cases have been gathered at the Hospital of the Salpêtrière by M. Charcot and by M. Vulpian, and have been, during the course of the year 1862, the object of important communications made by those physicians to the Society of Biology. In 1863, M. Cornil* had occasion to observe with M. Charcot some similar facts. He says that in six cases of old hemiplegia from cerebral lesion, they could discover five times the descending alteration in the mass of the brain, in the peduncle in the pons, in the anterior pyramid. He adds that they find the same alteration in the cord, but he does not fix either the seat or the extent of this alteration. The same year Leyden† published a remarkable case of secondary degeneration of the cord consecutive to compression by Pott's disease, and M. Cornil communicated to the medical society of observation an analogous case found in the service of

* A typographical error makes it appear that M. Cornil said that the secondary lesion occupied the anterior pyramid of the side opposite to that of the cerebral lesion. It is on the contrary always on the side of the diseased hemisphere that we find the pyramid altered.

Cornil. Note sur les lésions des nerfs et des muscles liées a la contracture tardive et permanente des membres dans les hémiplégies. (Société de Biologie, 1863.)

† Die graue degeneration der hinteren Rückinmarcksstraenge: Berlin, 1863.

M. le Dr. Charcot. In 1864, M. Laucereaux* compared the atrophy of the optic nerves in cerebral amaurosis to these descending degenerations of the cord, of which he gave some new observations. I had also at the same time an opportunity to meet frequently with these secondary lesions as a consequence of diseases of the brain or of primitive lesions of the cord, in the service of M. Charcot. I communicated several cases of them to the Anatomical Society and to the Society of Biology. Some of these observations have been published.† I gathered, in 1865, some new cases at the St. Eugênie Hospital in the service of M. Triboulet; and two cases to which I shall have to return, have been presented at the Society of Biologie, one by M. Cornil, the other by M. Charcot.

PATHOLOGICAL ANATOMY.

The secondary alterations of the spinal cord are never observed except in the fasciculi of the white substance. The gray substance has always been found intact. We could, indeed, foresee that this degeneration would only affect the conducting elements, and that it could not attack the elements which possess in a higher degree a peculiar activity, nutritive and functional, and which we

*De l'Amaurose liée à la dégénerescence des nerfs optiques dans les cas d'altération des hemisphéres cérébraux (Archives gen. de Méd., t. 1, p. 47: 1864.)

†Bouchard, Rapport sur une observation de compression de la moelle. (Bulletins de la société anatomiques, juillet, 1864.)

Aphasie sans lésion de la troisieme circonvolution frontale gauche. (Compte rendu de la société de biologie, 1864, p. 111.)

De l'Ataxie locomotrice progressive au point de vue de ses lésions anatomiques et de ses rapports avec diverses maladies peu connues de la moelle épinière. (Journal de Médecine de Lyon, Novembre, 1864.

Trousseau, Clinique Médicale de l'Hôtel Dieu de Paris, t. 11, p. 604: 1865, observation de Egris Valentine.

rightly consider as nervous centres. These degenerations have been observed in the white substance of the anterior part of the cord and in that of the posterior part. When a white fibre is wounded by whatever cause, in one part of its course, either in the cord itself or in its encephalic prolongations, the secondary alteration supervenes as a rule, only on one side of the primitive lesion, either above, or below, but it extends throughout the whole length of this portion to its central or peripheral extremity. Hence, the names of ascending degeneration and descending degeneration. Only the injured fibres are altered, and they are altered in their whole extent from the point of the lesion. Now as the white fasciculi of the cord receive on their way new fibres which cannot participate in the degeneration, it results that the secondary alteration will undergo a relative diminution according as we go further from the point primitively affected. As, on the other hand, the fibres which exist at this point, and degenerate, have not all the same destination, but are lost from time to time in the gray substance, it also results that, in following the degeneration from its origin to its termination, we will find that it undergoes a diminution not only relative, but even absolute. Let us see now in what this degeneration consists, and by what characters we can recognize it.

Here is the place to recall briefly the effects produced by the section of nerves. These experimental studies have given results which offer the greatest analogy with those which we are about to study. We know since the works of Waller* and of MM. Philippeaux and Vulpian† that the nerves, the connexions of which with the nervous centres are destroyed, present at the end of

* Loc. cit.

† Loc. cit.

twenty-four hours, a diminution of their peculiar excitability. This enfeeblement extends from the centre to the periphery, gradually augments, and finally all activity entirely disappears by the end of the fourth day. At this period the elements of the nerves which had not yet offered any appreciable alteration of their structure, commence to present marked modifications which bear witness to a serious disturbance produced in their nutrition. After the fifth day the medullary substance of the tubes is, so to speak, coagulated, and at the same time fissures form in its thickness and divide it into unequal and irregular blocks; this is what we call the segmentation of the nerve tubes. Very soon afterwards, these fragments of medullary substance are seen to be studded with fatty granulations, which go on multiplying, and which take the place of the former throughout the whole length of the tube. At the end of a certain time these fatty granulations are reabsorbed, and we see only the neurilema withered and shrivelled upon itself. What becomes of the axis-cylinder while this retrograde work is going on? This is a point which has not yet been completely cleared up. Let us add that the tubes thus destroyed can become regenerated; but they do not reform *in situ* but are only the expansion, the budding forth of tubes which remain healthy above the point cut.* It is at the end of fifteen days, at the soonest, that we can find the first indications of excitability in the regenerated nerve; at first in the parts nearest to the section, then at points successively more and more distant.

* This opinion, which appears generally admitted by physiologists, is not absolute. M. Vulpian has shown, indeed, that in certain animals, by choosing young subjects, we can obtain, without any influence of the centres regenerations of the hypoglossal and of the lingual nerve.

Similar researches would be applicable to secondary degenerations of the nervous centres; but experiments have not as yet, so far as I know, been employed in this special study. Besides, the pathological facts do not allow us to follow so easily the evolution of this morbid process. The cerebral or spinal disease kills the patient before the alteration of the involved tubes can become evident, or on the other hand death comes when the degeneration is complete, or when the fatty granulations have already been reabsorbed. I have nevertheless, twice made observations during the passage from the state of segmentation to the granular condition. I ought to mention here that, if we can come to conclusions from the analogy of what is known of the nerves to that which ought to take place in the spinal fasciculi, it is best to do so with an extreme reserve, for the facts known up to this day, and those which I have observed, indicate that the same alterations present in these two classes of organs marked differences, above all as regards their course and terminations. The white tissue of the cord seems to be much more delicate than that of the nerves; and a compression which, applied to a nerve, would pass unperceived and would be, at any rate, incapable of altering its structure, suffices to produce in the spinal cord a secondary degeneration. Besides the work of destruction of the tubes requires in the cord much more time than in the nerves; the granular condition lasts a much longer time; and finally if the regeneration of medullary fasciculi is possible, as I think it is, it advances much more slowly than that of the nerves.

L. Türcke, who seems to have been the first to submit secondary degenerations to a microscopic examination, has not given the details relative to the condition of the nerve tubes. He only points out the presence of a

great number of granular bodies and free molecular granulations in the diseased tissue which are thus transformed *in situ*. We cannot adopt this manner of seeing it, and we think that we have, at least at the commencement, not a disease of tissue but an alteration of element. In two cases of recent compression of the cord (one of a duration of fifteen days and the other of six weeks) I was able to see, by examining the degenerated fasciculi in a fresh condition, that a certain number of tubes were clearly in a state of segmentation; fissure, more or less deep, divided the neurilema transversely. At certain points of the preparation, these fragments were infiltrated with fatty granulations; others had been already partly destroyed by the retrograde work which gave the tubes a separated and notched appearance. Independently of the fatty granulations contained in the altered tubes, a large number were free among the elements, and at certain points they were agglomerated in a mass, in such a manner as to form what are known under the name of the granular corpuscles of Gluge.

In these cases, a fine section, made perpendicular to the axis after a maceration of some hours in alcohol, showed in the diseased fasciculi a large number of granular bodies scattered about under the form of black spots. All about these collections, the tissue had a healthy appearance, but the vessels presented on their surface lines of fatty molecular granulations or even a complete envelope which rendered them black and opaque under the microscope. In more advanced cases, where there were descending alterations, as a result of cerebral softening dating from six months to one or two years, I found, by lacerating the tissue of the diseased fasciculi while in a fresh condition, that the nerve tubes were healthy or slightly varicose, and that they were separated from one another by a small quantity of amorphous material,

which was usually soft, transparent and, as it were, gelatinous, enclosing numerous fatty granulations, granular corpuscles in greater or less number, and also nuclei analogous to those which exist normally in the gray substance of the nervous centres, and which M. Robin has described under the name of myélocytes. These nuclei were never very numerous; but they were the more abundant in proportion as the alteration was of older date. The appearance of the sections made after some hours' maceration in alcohol, did not materially differ from that which I have described for the first cases. We saw in the midst of a tissue which appeared almost healthy, granular bodies in variable numbers, and the vessels had a more or less atheromatous appearance. But the sections obtained from the same cord, after a maceration of some weeks in a weak solution of chromic acid, showed a notable difference between the healthy and diseased parts. Examined under a low power, the preparation showed in the altered portions bright striæ or transparent points disseminated between the tubes which, by their opacity, contrasted with the spaces occupied by the amorphous substance described above, and which alone allowed the light to pass easily. In the normal parts, the tubes pressed against one another, everywhere resisted the passage of light, and gave a uniform and sombre shade to the section.

Finally, in a case of compression of the cord, dating back for thirteen years, an examination made in the fresh state showed in the diseased fasciculi a soft, transparent and abundant amorphous matter, studded with myélocytes; and in the midst of it a few tubes, non-granular, but varicose. The fatty molecules were few in number, the granular corpuscles were found only here and there, and the vessels were almost normal. The fine sections made after maceration in chromic acid, and examined

with a low power, at once made evident the diseased parts, which contrasted in the clearest manner, by their transparency, with the healthy parts, which preserved their uniform dark tint. Besides, in the altered parts we saw scattered about in the transparent amorphous substance black dots representing the sections of tubes which had not been destroyed. I should add that the connective tissue which had thus taken the place of the nerve tubes had produced, owing to the contractility with which it is endowed, a particular deformity of the cord consisting in a longitudinal depression of the surface at the points nearest to the altered fasciculus.

By comparing these different alterations, and by considering their chronological succession, we can, I think, determine the morbid process in secondary degenerations.

Three principal facts arise from the preceding statement: 1st, the atheromatous appearance of the capillaries and the formation of granular corpuscles in the tissue which has degenerated; 2nd, the alteration, and afterwards the disappearance, of a greater or less number of the nerve tubes; 3rd, the formation of a connective tissue which takes the place of the tubes. Only two interpretations seem to me possible. We may suppose that an irritation produced at the point of the primitive lesion is propagated with rapidity through the whole length of the injured fasciculus, but in one direction only, viz.: that of its physiological conductibility; that there hence results a slow inflammation, exactly limited to the parts whose functional activity is no longer called in use, and which cannot spread to contiguous parts; and that this inflammation gives rise to a morbid production of connective tissue which unites the nerve tubes, vitiates their nutrition, and at last causes their disappearance.

Under this hypothesis we might, with M. Robin,

consider the corpuscles of Gluge as leucocytes which have undergone the granulo-fatty alteration.* Under another hypothesis which I hope to make more worthy of acceptance, the tubes injured in one point of their course are primarily altered in all that portion which has lost its connection with their centre of origin, without there being any trouble of nutrition in the tissues which they pass through. Every thing then goes on as it does in the peripheral end of a divided nerve. The substance of the tubes is changed into fatty granules which are diffused through the tissue, some isolated, others accumulated in masses, (granular corpuscles,†) or in lines along the vessels, (atheromatous appearance of the capillaries.) The nutritive activity of the tissue is stimulated to action by this foreign substance which infiltrates it, absorption commences and causes it to disappear little by little, while at the same time proliferation of the connective tissue fills up the vacancies.

The first hypothesis seems to me liable to very grave objections. Independently of the fact that there is something strange in imagining an inflammation which should develope itself on one side only of the primary lesion, and always on the same side, which should spread rapidly through the whole length of a spinal fasciculus without manifesting itself by any symptom, and which, in this sudden extension, should limit itself to a very narrow band without encroaching upon contiguous parts, which notwithstanding have intimate nutritive relations with the diseased parts, by their nervous and vascular connection, we should suppose that the microscope should

* It is seldom, except in inflammatory softening, that we can attribute to leucocytes the origin of granular corpuscles.

† It may be that certain granular corpuscles are the result of the granulo-fatty transformation of drops of the white substance of Schwann.

show proliferation of the connective tissue before showing the alteration of the tubes. Now this is contrary to what we observe. The first lesions which we see are the segmentation and the retrograde alteration of the tubes; the morbid production of connective tissue is secondary, and occurs much later.

On the other hand, if the increase of connective tissue was the initial phenomenon causing as its consequence the disappearance of the tubes, secondary degeneration would be nothing more than a sclerosis, and should have the character of sclerosis. We know that, in this latter affection, the proliferation of connective tissue strangles the nervous elements, vitiates their nutrition, and causes their atrophy and disappearance; but this disappearance has an entirely special character: the white substance of Schwann diminishes in thickness, generally in an unequal manner, so as to give to the tubes a varicose appearance; this substance may be entirely absorbed in some parts where the axis-cylinder remains bare; we can then trace it to a point where it disappears in a sheath of medullary substance still intact; then it reappears further on; finally the absorption of the medullary matter of the tubes becomes complete, and we can see the axis-cylinders remaining in the midst of the sclerosed mass, parallel to one another, and similar in appearance to the fibres of subcutaneous cellular tissue, from which they are easily distinguished by the action of certain re-agents. Then, there is direct atrophy of the medullary substance of the tubes; in secondary degenerations there is no atrophy of this substance; it disappears by the necessary intermediation of a process entirely different from atrophy, by a retrograde transformation, and, before being absorbed, it loses its usual appearance, its cohesion, and even its chemical constitution. I may add, that I have never been able to find

the denuded axis-cylinders in the case of secondary degeneration, and no observer has remarked their persistence, which we often find in cases of sclerosis. Finally, a new differential character is, that the sclerosed tissue is almost always studded with a considerable number of amylaceous bodies; these bodies are totally absent, or only exist in small quantities in parts attacked by secondary degeneration.

It is not that I do not comprehend the analogy which exists between the proliferation of connective tissue which is observed at an advanced period of secondary degenerations, and the new morbid product which primitively constitutes sclerosis; but we will see what essentially different parts this hypergenesis takes, in the two cases, with regard to the disappearance of the nerve tubes.

Here is the place to make an important remark: in a nerve attacked by secondary degeneration, all the tubes are not altered; some preserve their structure and their functions almost intact; these are those which, already existing at the part primarily injured, have been respected by the injury, or those which have emerged from the gray substance beyond the primary seat of alteration. These tubes, after the disappearance of those which normally surround them, become isolated in the midst of the connective tissue which is undergoing proliferation, and which by its contact cannot fail to modify their vitality. In fact they become varicose, just as is seen in sclerosis. It is on account of these considerations that I have, in a former work,* described secondary degenerations under the name of secondary

* De l'Alāxie locomotrice progressive au point de vue de ses lesions anatomiques et de ses rapports avec diverses maladies peu connues de la moelle épinière. (Journal de Medicine de Lyon, Novembre, 1864.)

sclerosis or false sclerosis, in contradistinction to primitive sclerosis or true sclerosis.

To finish with the processes of secondary degenerations, I ought yet to say a few words about the vascular alterations observed in the diseased tissue. I have already pointed out the atheromatous appearance of the capillaries; it is observed almost constantly, but in very variable degrees, and this fact, added to other characters indicated further on, seem to justify the comparison which M. Gubler* has made between secondary degeneration and chronic cerebral softening. The yellow, depressed patches which are so often met with on the surface of the brains of old persons, present in a peculiar degree all the histological alterations which we have described, but in a much more marked form. It is not probable that the morbid process, although producing similar results, is the same in the two cases. Although pathological anatomy and experiment have thrown light upon certain points of the pathogeny of cerebral softening, the mode of formation of these yellow patches is far from being understood. As for the atheromatous condition of the capillaries which is found so constantly and to so great an extent in this affection, we know not whether it is the product or the cause of the disease. In secondary degeneration, this condition of the vessels seems to me to be consecutive to the alteration of the surrounding tissues.

And first, it is not the result of senile degeneration, for I have succeeded in finding it in a very advanced degree in very young children, as a result of compression of the cord by Pott's disease.† The fatty condition

* Loc. cit. (Archives gén. de Med., t. 11, p. 31: 1859.

†I should protect myself against the charge which might be brought against me of having described, as atheroma of the capillaries in children, a condition of the vessels of the brain usual at

of the capillaries is only the consequence and the index of an alteration of nutrition of that portion of the tissue which has lost its physiological activity; therefore it is only the indirect consequence. I could with difficulty comprehend that a capillary vessel should become fatty only because the neighboring nerve tubes do not perform their functions; but if the neighboring nerve tubes are greatly modified in their structure, it may be that the abnormal materials of disassimilation which they give up to the capillaries, may produce a secondary trouble in the vitality and structure of the latter. Besides, the alteration with which we are occupied, does not seem to me to have advanced to this degree. If I am not deceived, there is only an atheromatous appearance, and not a fatty transformation of their substance. During the early period of secondary degeneration in young subjects, the abundant fatty granulations appear to me to envelope the vessels rather than infiltrate them; they seem to be external to the membrane proper of the capillary vessel, which latter does not appear to be perceptibly modified. I have even demonstrated in several cases that they were accumulated between that membrane proper and its envelope of connective tissue, in that intermediate space to which M. Robin has called attention. I do not deny that these facts should be examined into: for the interpretation which I propose is founded upon only a limited number of cases. At all events it seems to me to give a satisfactory explanation of the facts observed,

that period of life, a condition characterized by the presence along the capillaries of little refracting beads and granulations which are evidently situated in the lymphatic interspaces described by M. Robin, and which, by their disposition in lines or their accumulation at certain points, principally in the angles of bifurcation, may resemble in a superficial examination, the atheromatous alteration.

and of their evolution, since the atheromatous appearance, very marked at a certain period, seems afterwards to diminish, so that when the connective tissue is completely formed, the granular corpuscles and molecular granulations have in a great measure disappeared from the diseased part. This theory of the atheromatous alteration of the capillaries of the nervous centres could not be peculiar to secondary degenerations, for it enters into that general law expressed by Billroth, as follows:* "The fatty degeneration of the capillaries of the brain, or rather their envelopment in fat, is the consequence not the cause, of a defect of nutrition in the central nervous tissue."

One could object to the theory which I have just given of the primitive granular fatty degeneration of the nerve tubes of the cord that, in nerves where this alteration has been studied to better advantage, the observed phenomena differ materially from those which I have described above. This is because, in the nerves, each primitive fibre is contained in a solid and resisting envelope, which is wanting to the tubes in the nervous centres. In the nerves, the fatty granules resulting from the transformation of the substance of Schwann remain imprisoned in this envelope, and cannot distribute themselves in the surrounding tissue as they do in the cord. This anatomical peculiarity seems to me sufficient to explain the differences of appearance which the same alteration presents according as it is in one tissue or another.

It would still remain to study the regeneration of nerve tubes in the spinal fasciculi which recover their

* Theodor Billroth, Ueber eine eigenthümliche gelatinöse Degeneration du Kleinhirnrinde nebst einigen Bemerkungen über die Beziehungen der Gefässerkrankungen zur kronischeu Encephalitis (Archiv-der Heilkunde Dritter Jahrgang, p. 47.)

functions after having been affected by secondary degeneration. But the materials for this study are completely wanting; and, in a general way, we may say that the regeneration of spinal fibres has never been established by any direct observation. On one occasion, MM. Charcot and Vulpian thought they preceived the traces of reproduction of the nerve tubes in a case of sclerosis of the posterior columns. This appearance has never even been mentioned among the results of secondary degenerations; and if I said above that I believed in the regeneration of the nerves of the cord after these alterations, it was because I relied upon clinical considerations which I shall treat of hereafter.

I should now point out the methods of investigation applicable to anatomical study of secondary degenerations.

Direct inspection often allows us to seize upon certain peculiarities which may put us upon the track of this alteration. Thus in descending degenerations, a result of some old brain affection, it is not uncommon to find the peduncle of the diseased side smaller than that of the other; we then remark, after having removed it envelopes, that its color is changed, it presents on its inferior aspect a line of a yellowish gray in the course of its fibres, of greater or less size, situated sometimes at its internal, sometimes at its middle and sometimes at its external part, according to the location which the primitive alteration occupies in the hemisphere. In these cases it is not uncommon for the pons to present a more or less marked flattening on the same side. The medulla oblongata, deprived of its membranes, also shows a marked difference between the two anterior pyramids. The pyramid of the diseased side is, like the peduncle, small and yellowish. This atrophy of the pyramid renders the olivary body of the same side

more protuberant, and might lead us to think there was a disease of this organ.

As a general rule, the consistency of the degenerated parts is not changed; but in one case M. Gubler noticed a softening of this tissue.

As for the cord, an external examination rarely furnishes any indications, unless it be in very extensive and very old alterations of a hemisphere, and more particularly in cases of cerebral agenesis. We then find a diminution in size of that half of the cord opposite to the diseased hemisphere. In cases of the same kind, if the atrophy of one half of the cord is not evident, we observe sometimes a slight deformity of the organ consisting in a longitudinal depression in one of its lateral parts, a little in advance of the posterior roots. This lesion, however, cannot be well traced unless the cord has been previously hardened.

Seen through its membranes, the cord does not present any modification of color at the situation of the altered fasciculi; even when the membranes are detached without affecting its tissue, its color appears to be normal. But, if we make a section of the organ in a direction perpendicular to its axis, we can often see that certain portions of the white columns have not the same appearance as the healthy parts. It is sometimes a yellowish gray tint, sometimes a semi-transparent, bluish gray tint, like that of milk diffused in water; and sometimes again it has the gelatinous grayish coloration of sclerosis. The yellowish color is principally observed in cases of degeneration with an abundance of granular bodies, that is to say in cases of not very long standing. Nevertheless, and I should insist upon this point, an examination of the diseased tissue of the cord with the naked eye is usually incapable of leading us to suspect even quite marked alterations.

After this preliminary inspection, and before submitting the cord to any preparation, it is best to examine with the microscope some portions of its tissue, removed with small curved scissors, from the parts supposed to be diseased. These fragments teased out in a drop of water, should be examined with a power of from 150 to 300 diameters. We thus recognize the condition of the tubes, the atheromatous appearance of the capillaries, the granular corpuscles, the molecular granulations and the starch bodies. We then replace the water of the first preparation by a few drops of a weak solution of carmine whose alkalinity has previously been neutralized by acetic acid. The amorphous matter interposed between the tubes then becomes plainer, nuclei appear, and the capillaries are made more visible. All these details may be made still more precise by the addition of a drop of acetic acid, after having taken the precaution to remove the excess of carmine by washing with pure water. It is often more expeditious to replace the carmine by an aqueous solution of with or without the addition of acetic acid.

After these primary investigations, the cord is placed in alcohol at 36° C., and after a few hours it will have accquired a sufficient degree of firmness to enable us to make quite thin sections perpendicularly to its surface. By treating these sections with acetic acid, compressing them slightly between two glass slides, we can ascertain, with a power of from 80 to 120 diameters, at what precise points the granular corpuscles and atheromatous vessels are situated. This preparation cannot give other indications; but we can renew the first examinations which I have mentioned above, on the portions of the cord thus treated with alcohol; the nerve tubes then have sharper outlines, and the observation is less impeded by the beads of the white substance of Schwann

which are constantly formed in preparations made with the fresh cord.

The cord is then placed in a solution of chromic acid, and, by the end of two or three weeks it will usually have acquired a sufficient degree of firmness. Then it is not very rare for the greenish yellow color of its section to present, at the points of degeneration, a lighter tint than at the healthy parts; and we can often map out exactly the space occupied by the degeneration, by pouring upon the surface of the section a few drops of a concentrated solution of carmine. By washing this surface, at the end of a minute, with a camel's hair pencil dipped in water, the diseased parts only remain colored with a lighter or darker tint of violet, according as the connective tissue of new formation is more or less abundant.

The examinations of these sections show also the deformities which the cord may have undergone, and allow us to measure very exactly the dimensions of each fasciculus, by appreciating the differences which may exist on one side or the other.

Finally it is best to remove from these hardened cords some very thin slices which, treated first by caustic soda, then by glycerine, show in the clearest manner, even to the naked eye, the altered parts. The latter appear as clear, transparent patches, plainly contrasting with the surrounding tissue which remains opaque; and under the microscope, they seem studded with a variable number of black points, representing the tubes which have not been attacked by the degeneration.

If we wish a preparation of the whole structure which will also serve for a study of the elementary alterations in detail, the section must first be placed in a weak solution of carmine, made neutral by acetic acid; at the end of a few hours, when we consider it sufficiently colored,

we wash it with water, then with absolute alcohol, finally we treat it with rectified essence of turpentine, and finish the preparation in Canada balsam.

This mode of preparation is also applicable to longitudinal sections. We see in them the varicose condition of the tubes, which, although comprised in the diseased parts, have been respected by the degeneration, but have been surrounded and then deformed by the connective tissue of new formation.

Thus far, I have studied the secondary degenerations in themselves, without considering the varieties of situation which these alterations may present in the different columns of the cord. The anatomical facts are general; they are applicable to all cases, whatever be the direction in which the degeneration is produced, ascending or descending, whatever be the nature or location of the primitive lesion to which they succeed.

I am now going to commence the especial study of these degenerations, and demonstrate in what direction these lesions are produced, to what columns and what portions of columns they are limited, according as the primary disease is situated in such or such a part of the nervous system. I shall study successively the secondary degenerations of the spinal cord, in consequence of primitive lesions, 1st, of the cerebral hemispheres; 2d, of the cerebral peduncles; 3d, of the pons; 4th, of the medulla oblongata; 5th, of the cord itself; 6th, of the spinal roots. At a future time we shall without doubt form a seventh class for ascending degenerations of the cord in consequence of primitive lesions of the ganglia of the posterior roots; but I do not know that any observation is in existence at this day which can enter into that division.

I. SECONDARY DEGENERATIONS AS A RESULT OF PRIMITIVE LESIONS OF THE CEREBRAL HEMISPHERES.

These degenerations are the first which were discovered. It is to these that the observation given in the Sepulcretum refers; these are those the existence of which M. Cruveilhier suspected; it is these which are referred to in the first exact works published upon this subject by L. Türcke, by MM. Charcot and Turner, and by Schroeder Van de Kolk. Up to this time a certain number of observers have proved facts of this kind; but we may say that the subject, as yet very incompletely known in an anatomical, is not at all understood in a clinical point of view.

We will point out hereafter the symptomatic peculiarities which may be referred to these degenerations. As to the precise seat of the alteration, the numerous facts which we have gathered during the last three years, both at the Salpêtrière, in the service of M. Charcot, or at the hospital Sainte-Eugénie, in wards of M. Triboulet, seem to us to fully confirm the ideas advanced in 1851, by L. Türcke.

But before entering into the details of this anatomical study, some preliminary questions should be decided.

And first, Do all morbid conditions of the hemispheres determine secondary degenerations? Thus proposed, the question ought to be answered in the negative. I have never found a trace of this alteration in simple cases of compression by tumors of the membranes, by effusions into the arachnoid, or by thick false membranes of the dura mater, with meningeal hemorrhages. Even very extensive superficial lesions of the convolutions do not produce any descending degeneration. This lesion is met with, neither in acute meningeal encephalitis, nor in tuberculous meningitis, nor in the diffused meningeal encephalitis of general paralysis, nor in the majority of

superficial softenings of the convolutions either red or yellow. But certain lesions of the peripheral portions of the brain which interest the deep layers of the gray cortical substance, and destroy them, such as the yellow patches, and which encroach even upon the subjacent white tissue, may give rise to secondary degenerations which are usually not very marked.* As a general rule these degenerations result from lesions of the central portions of the hemispheres. It is above all in cases of hemorrhages or of softening of the corpora striata that they easily reveal themselves to the observer. Still it is not necessary that the lesion should occupy exclusively the centre of the ganglia of gray substance. There is a remark verified a number of times by M. Charcot and M. Vulpian, that the most manifest secondary degenerations succeed primary lesions which have destroyed to a greater or less extent the little white bands† interposed between the two corpora striata (capsule interne de Burdach.) Lesions of the optic thalami also produce descending degenerations, which however are generally less marked than those which are consecutive to destruction of the corpora striata. Finally, I have observed recently, with M. Charcot, a case of secondary alteration of the cord connected with the existence of depots of cellular infiltration in the centrum ovale. We see that a certain degree of vagueness reigns over this subject, and that the intensity of secondary degeneration, compared with the extent of the primitive lesion and with the exact situation of this lesion, deserves to be studied

* See the observation already cited of Egris Valentine, where I have seen evident descending degeneration result from a yellow patch spread over several convolutions, with integrity of the corpora striata and the optic thalami, as well as of the expansions of the peduncles. (Troussean, Clinique Medic., loc cit.)

† Taenia semi-circularis.

in a more precise manner. This will be the surest way of knowing what relative quantity of nerve tubes each part of the brain sends directly to the spinal cord, and what location these tubes occupy in the substance of the rachidian axis.

Thus far, the labors of M. Türcke have only determined with exactitude the distribution of the descending degeneration in consequence of cerebral lesions; and if the results at which he has arrived are incomplete, as much in regard to the process of this degeneration as to that of the final evolution which it undergoes, we ought to acknowledge that he has determined with certainty the location which the lesion occupies in the cord. More recent works have added nothing to his description, and the numerous facts which I have been able to collect, have all conformed to the localization which he had indicated. A single fact anterior to his researches bore witness to the correlation which may exist between atrophies of the cord and lesions of the brain; but this fact had not been understood. I would speak of the observation of Wepfer, which I have mentioned before. It concerned a young girl with paralysis of the left arm, who had in the right hemisphere two cavities as large as eggs and filled with a turbid liquid. The corpus striatum and a portion of the corpus callosum were ulcerated. It is said, in the observation that the cord did not fill the rachidian cavity which contained a good deal of sanguinolent serum. The author adds: "*Quae* (the cord) *firma et nitida erat, sinistra tamen pars dextra minor videbatur.*" The history of secondary degenerations of the cord was limited to this phrase, before the works of Türcke. However, Rokitansky* had remarked, after a considerable loss of substance of the hemispheres by hemorrhage or inflammation, an atrophy

* Patholog. Anat., 1re edit., t. e, p. 715.

of the peduncle, of the pons Varolii, of the medulla oblongata and of the spinal cord.

Thus, there exists an intermediate step between the alteration of the cord and the primitive lesion of the hemisphere. The degeneration is above all marked in the peduncle and in the medulla oblongata. It was that which attracted the attention of M. Cruveilhier, and that which he has described with exactitude. I should say that the succinct description which he has given of it is a resumé of a long observation of facts which have not been published. A single case of descending degeneration as the result of lesion of one hemisphere is given in his Atlas of pathological anatomy;* it is so incomplete that it could not have enabled this author to give the excellent description of secondary degenerations of the medulla oblongata to which I have already referred. The observation to which I allude is that of Jeanne Hamel, who died at the Salpêtrière, the third of January, 1833, at the age of 72 years. This woman had a left incomplete hemiplegia, a paraplegia with rigidity, and showed some defects of intellect. Death was the result of an acute red softening of the convolution. They found in the midst of the cerebral substance several little cicatrices of former effusions and linear induration in the substance of the left peduncle. The pons was a little deformed, the median line prominent, the lateral portions depressed; it contained in its substance, on the right side, a little cellular cavity; the cord was indurated. We might ask if any one of these lesions is the result of a secondary degeneration. The induration of the left peduncle could not be considered as a descending alteration, from the fact that it was indurated; besides it is not stated whether there were marks of disease in the

* Anatomie Patholog., 32 livrais., p. 15.

left hemisphere; finally in this case the hemiplegia should have been in the right and not in the left side. The cellular cavity of the pons was only the trace of a primary softening. The flattening of the pons on the left side could, alone, be attributed to a secondary degeneration of its longitudinal fibres; but did this degeneration have its primitive cause in the hemisphere or in the peduncle? As for the cord, its induration, which was indicated by the paraplegia, could not in any manner be considered as the result of a descending alteration.

I have already indicated the characters of descending degeneration in the medulla oblongata. As this is not the object of this work, I will not here enter into fuller details, and I pass to the examination of the situation which the secondary degeneration consecutive to lesions of the hemispheres occupies in the spinal cord.

The alteration which, in the bulb, is limited to the anterior pyramid of the side corresponding to the primary lesion of the brain, penetrates the cord following the distribution which normal anatomy indicates; it follows the decussation of the pyramids, and occupies in the cord the side opposite to the primitive lesion. In all cases where a secondary alteration of the bulb is manifest we find an alteration of the same nature in the antero-lateral column of the cord on the opposite side. But this alteration does not spread throughout the whole thickness of the antero-lateral column; it occupies a precise location in it, it is limited to the posterior portion of the lateral column between the postero-lateral fissure and the ligamentum denticulatum. It is at this point that Türcke found the granular corpuscles; it is there also that the lesion has always seemed to me to be located. In cases of considerable and very advanced degeneration, where a large number of tubes have dis-

appeared and have been replaced by connective tissue, we see the antero-lateral column, healthy in the remainder of its structure, present a spot as if colored by carmine at this exact point which in their sections contrasts by its transparency with the surrounding tissue, and looks like a hole cut by a punch. All around the medullary is normal and we always find a little band of healthy white substance which separates the altered portion from the pia mater. This is the reason why even in cases of very considerable degeneration the external examination of the cord does not allow us to perceive any modification in the color of the altered column, while we can do so in true sclerosis of the lateral columns when the diseased tissue, being in direct contact with the meninges, is recognizable from its peculiar gray tint. However, in hardened cords, we can sometimes see at this point a deformity of the organ, a depression of its surface which forms an abnormal furrow in advance of the line of attachment of the posterior roots. The alteration of the posterior portion of the lateral column, more marked in the cervical region, diminishes as it recedes from the bulb; but we can in most instances follow it throughout a great extent of the cord, sometimes even to the inferior part of the lumbar enlargement.

We know that at the inferior part of the bulb the decussation of the pyramids is not complete, but that a portion of the fibres which compose each pyramid approach the median line so as to form the internal part of the anterior column of the same side. This anatomical fact would lead us to expect that in certain secondary degenerations we should observe, as a result of a lesion of a single hemisphere, a degeneration of the posterior part of the opposite lateral column, and of the internal part of the corresponding anterior column; that

is to say a lesion of both sides of the cord. This is what we see in reality. Out of six cases of secondary degeneration consecutive to lesions of the brain, L. Türck has seen, in three, the alteration of the internal part of the anterior column accompanying that of the lateral column of the opposite side. I have had occasion to observe this double degeneration with M. Charcot; but, in a greater number of autopsies, I have only once been able to see the condition of sclerosis which succeeds to the destruction of the tubes, in the anterior column of the side of the cerebral lesion.* In this case the alteration formed a narrow little band, clearly distinct from the healthy tissue, applied to the expansion which the membranes send into the anterior sulcus, and reaching in front, the inner surface of the pia mater, and behind, the anterior face of the commissure. Perhaps my attention has not been sufficiently fixed upon this question; at all events, this alteration of the anterior column seems to me to be rare, besides it is always accompanied by an alteration of the same nature, and more pronounced, of the posterior portion of the lateral column of the opposite side. In order that it should occur, the alteration of the anterior pyramid must be extensive and must interest its external portion. Secondary degeneration of the internal portion of the anterior columns, as a result of lesions of the hemispheres, does not extend through the whole length of the cord. In the case which I have seen, it could not be recognizedat the middle of the dorsal region; but Türck says, that, in two cases, he has been able to find the granular corpuscles as far as the level of insertion of the roots of the last intercostal nerves.

* I am indebted to MM. Charcot and Vulpian for the communication of an analogous observation gathered by them in 1862.

II. SECONDARY DEGENERATIONS AS A RESULT OF PRIMITIVE LESIONS OF THE CEREBRAL PEDUNCLES.

The absence of proper documents will oblige us to make this a brief chapter. A single observation has been published up to this day; still it may be disputed whether it was secondary degeneration. It had reference to a fibrous tumor of the left cerebral peduncle in an epileptic. The case was presented to the Society of Biology by MM. Cornil and Thomas.* The tumor had produced an atrophy of the peduncle, and this atrophy extended to the pons and to the anterior pyramid of the same side. The tissue of the atrophied parts presented no analogy to that of the tumor; it was in every respect similar to that of parts affected by sclerosis, the posterior columns of those suffering from ataxy, for example; no granular corpuscles were met with. It may be that this was not a secondary degeneration at the period of proliferation of connective tissue, but a sort of chronic inflammation of the peduncle occasioned by the tumor, an inflammation which would be propagated in the direction of the fibres of the part, just as is usually observed in primary sclerosis. At all events, the condition of the cord has not been given in the observation.

I have recently seen, in an autopsy made at the Salpêtrière, two symmetrical points of softening in the cerebral peduncles, the pons was flattened on each side of the median line, and the anterior pyramids presented the atrophy and the yellowish gray color characteristic of secondary degeneration; but a microscopic examination was not made, and the cord was not examined.

III. SECONDARY DEGENERATIONS AS A RESULT OF PRIMITIVE LESIONS OF THE PONS.

Observations are still more completely wanting on this subject. A single case, although very incomplete,

* Comptes-rendus de la Societé de Biologie, 1864, p. 46.

seems to us worthy of being mentioned. I borrow it from the Atlas of Pathological Anatomy of M. Cruveilhier.* Marie Duffet, aged 57 years, died at the Salpêtrière, June 3, 1834. Was hemiplegic on the right side; motion was entirely lost on this side, and there was an incomplete loss of sensibility. The members of the left side were not possessed of their natural power of motion. At the autopsy the brain was found to be healthy; but in the pons was discovered an old apoplectic depôt implicating both sides, more extensive superficially on the right, but deeper on the left. The anterior pyramids were atrophied, especially the left.

The plate which represents this lesion being inexact, I cite word for word the correction of the author: "The anterior pyramids were atrophied to such a degree that, in my notes taken at the time of the examination, I have written: 'No anterior pyramid on the left side, the right anterior pyramid atrophied.' On this account I ought to correct the figure, which, having been finished in my absence, resembles the normal condition a great deal too much."

Finally it is said in the observation that the cord was healthy.

Although incomplete, this observation at least proves that the intensity of the secondary degeneration is the more pronounced as the primitive lesion is nearer the bulb.

IV. SECONDARY DEGENERATIONS AS A RESULT OF PRIMARY LESIONS OF THE BULB.

In proportion as the primitive lesion approaches the spinal cord, the secondary degeneration of this nervous centre ought to become more marked and more complicated: every deep lesion of the peduncle, of the pons or

* 21 livraison, pl. v., fig. 3.

of the bulb ought to produce an alteration not only of the tubes which have their origin in the altered part, but also of those which, commencing higher up, pass through that part and are there injured in one point of their course. This increasing complication of secondary degeneration could not have been treated of in the preceding paragraphs, since in none of the cases of primary alteration of the peduncles or of the pons Varolii which we have mentioned, has the condition of the cord been studied. It is not so in primary lesions of the bulb; but these cases are rare, for the alterations of this part of the nervous centres generally produce death before the degeneration has had time to become developed. We have not found in the authors any observation which can instruct us with regard to the disposition of descending degenerations consecutive to primary lesions of the rachidian bulb. The description which we will give of them rests solely upon two observations, of which one was taken by us at the hospital Sainte-Eugénie, the other was sent to us by M. Charcot.

The first case regarded a little girl of five years, affected by cervical arthritis. An abscess produced by caries of the axis softened in front of the dura mater, and lifted this membrane even in the interior of the cranium for two centimetres in front of the occipital foramen. The bulb was compressed and flattened from before backwards by this collection of fluid, and, besides, an inflammation, which, had caused adhesions anteriorly between the arachnoid and dura mater, had implicated even the tissue of the bulb, the superficial portion of which was the seat of a red inflammatory softening. Death, which was the result of this inflammation, occurred about fifteen days after the commencement of the paralytic symptoms which we could attribute to the compression. Several sections made at different parts of the cord at points

which were neither compressed nor inflamed, showed, in the fresh condition, numerous granular corpuscles throughout the whole of the antero-lateral columns accumulated especially in the posterior half of the lateral columns. The capillaries presented in a marked degree the atheromatous appearance; there was not as yet any proliferation of the the elements of connective tissue. The posterior columns were perfectly healthy.

In the second case, the bulb was compressed by a dry arthritis; the new formations of bone narrowed considerably the occipital foramen, and a marked thickening with increased length of the odontoid process diminished still more the free space occupied by the bulb. The compression was exerted especially upon the anterior and left lateral portion of this organ; the point most compressed appeared to be the inferior part of the left pyramid, immediately above the decussation. The commencement of trouble dated back one year, therefore there was an abundant production of connective tissue in place of portions of the cord secondarily degenerated. These anatomical specimens have been presented to the Society of Biology, by M. Charcot, and we owe to his kindness the opportunity of renewing upon this cord, preserved in chromic acid, the examination which had been made by him in the fresh condition, at the time of the autopsy, and afterwards upon thin sections, after hardening the organ.

In the fresh condition there was seen by the naked eye, thoughout the whole extent of the rachidian axis, a gray coloration of the posterior portion of the right lateral column, in the seat of election of secondary degenerations consecutive to cerebral lesions. This gray tissue was composed of varicose tubes, an amorphous transparent granular matter, numerous oval or spherical nuclei and amyloid bodies. The sections made from the

hardened cord showed at this point a diminution of the tubes; but this transparent spot due to the accumulation of connective tissue of new formation, did not resemble a simple hole, it reached the inner face of the pia mater; besides, the neoplastic production continued along the surface of the antero-lateral columns and penetrated into the anterior sulcus, thus showing a destruction of the more superficial tubes of these columns on both sides. Finally at the posterior portion of the left lateral column a rarefaction of the tubes could be seen as on the right side but less considerable. This degeneration of the left lateral column was not recognizable in sections made in the fresh condition. I should add that, on both sides, but above all on the right, the proliferation of connective tissue of the posterior and external portion of the lateral columns had implicated for a very short distance the contiguous portion of the posterior columns, between the extremity of the posterior cornua and the collateral posterior sulcus. These different lesions manifested the characters which I have pointed out, only in the cervical region; below the brachial enlargement, only the alteration of the lateral columns was visible, and at the inferior part of the dorsal region, only the degeneration of the posterior part of the right lateral column could be found, which was then seen with the same characters as those of degenerations of cerebral origin; that is to say that the transparent portion occupied by the connective tissue formed a circular hole separated from the meninges by a little band of healthy medullary tissue.

We see that in these two cases the seat of the lesion does not appear to be the same; however, we observe that the degeneration was not limited to the posterior parts of the lateral columns, but that it extended in the substance of the antero-lateral columns. The only

difference is that, in the recent case, we found some granular corpuscles in the substance of the anterior column and of the anterior part of the lateral column, while in the case of longer duration we did find that there was, in these parts, an accumulation of connective tissue which had undergone proliferation only at the surface. But we must remember that the new formation consecutive to destructions of tubes, becomes developed and is apparent only in those cases where a considerable number of nervous elements has been destroyed at the same point, while the degeneration of a few isolated tubes suffices to produce granular corpuscles. The alteration of the substance of the anterior portions of the antero-lateral columns may therefore be manifest in a recent case, and not be appreciable in a case of longer duration. This remark is applicable to a very large number of cases of secondary degenerations. It is very seldom that, in cases where death occurs during the first few months after the commencement of a very limited lesion of the brain, we do not find in the cord granular corpuscles or atheromatous capillaries; on the contrary it is often the case that we cannot find any proliferation of connective tissue at the seat of election in even more extensive cerebral lesions when death takes place at a period when the granular corpuscles have had time to disappear. The study of degenerations consecutive to primary lesions of the cord itself is about to furnish us new arguments in support of this mode of considering the subject.

I should still say a word or two about the very limited alteration noticed in the posterior columns of the cervical region in the last observation. Are there some tubes which arise from the bulb, and, following a downward course, occupy the most external part of the posterior columns? in other words, are there, in the

posterior columns, some whose centre of nutrition is situated higher up in the bulb or beyond it? All the known facts up to this day and those which we will discuss in the two following paragraphs contradict this hypothesis, and perhaps in this case we have an instance of the propagation to neighboring parts of a formative irritation, the seat of which was in the lateral columns.

In conclusion, we may say, after these two observations, that secondary alterations of the cord depending on primitive lesions of the bulb, affect the whole of the antero-lateral columns with a greater intensity at the surface than in the deeper parts, but that the degeneration implicates the greatest number of tubes in the posterior part of the lateral columns.

V. SECONDARY DEGENERATIONS CONSECUTIVE TO PRIMITIVE LESIONS OF THE SPINAL CORD.

The secondary degenerations of the cord which follow a lesion at a given point of this nervous centre have been observed under very varied circumstances; but it is more especially as a result of compressions of the cord by tumors of the meninges, by purulent collections in the rachidian canal, by fractures of the vetrebral column, and above all by Pott's disease, that they have been studied. They have also been seen to follow diseases of the tissue of the cord as partial scleroses; but these last facts, I should say, are still very obscure. If the original disease of the cord is acute, it seldom gives time for the degeneration to be produced; if on the other hand it is chronic, the alteration of the tubes, even in the locality of the lesion, advances slowly, and the portion which is external to this locality may then gradually become atrophied, in such a manner that the production of the secondary lesion does not appear to be absolutely identical with that which we have formerly indicated.

Every lesion of the cord at one point of its course destroys, by descending degeneration, not only the fibres which come directly from the different parts of the brain, but also those which have arisen from the gray substance of the cord above or at the level of the part injured. The descending degeneration then presents the greatest complications. On the other hand, the posterior columns, injured at one point of their course, degenerate on one side of the point primitively altered. This alteration of tubes whose centre of nutrition is at their inferior extremity, gives rise to ascending degenerations which we will constantly find in the posterior columns, and sometimes in a certain part of the lateral columns.

I shall first study the descending alterations: they have the greatest analogy to those which result from primitive lesions of the bulb. This descending degeneration has been observed quite a large number of times by L. Türck. His first memoir* contains three cases of it; two years later he reported in detail twelve new cases.† In the majority of the cases it was connected with compressions of the cord by Pott's disease. Below the primary lesion, the posterior columns were always found perfectly normal; the degeneration affected exclusively the antero-lateral columns which were seen to be studded throughout their entire substance with granular bodies, accumulated principally at the posterior part of the lateral columns. Passing from the point primitively injured, and approaching the cauda equina, it was found that the alteration of the anterior columns, and of the anterior part of the lateral columns, dimin-

*Ueber secundäre Erkrankung, etc. (Comptes-rendus de l'Acad. des Sciences de Vienne, Mars, 1851.)

†Ueber secundäre, etc. (Comptes-rendus de l'Acad. des Sciences de Vienne, Juin, 1853.)

ished in intensity and disappeared entirely at about the fourth insertion of nerves below the point compressed; but, at this level the granular bodies still existed in abundance in the posterior and external part of the lateral columns, and in some cases could be found even at the inferior part of the cord.

Ten years later, Leyden reported a fine case of secondary degeneration of the cord by Pott's disease, in a little girl aged three years and nine months.* Below the point compressed the antero-lateral columns were the seat of a gray degeneration, especially at the periphery; the posterior columns were healthy. Leyden seems to me not to have understood the process of secondary degeneration which has reached the exaggerated production of connective tissue, and he was wrong when he wished to support upon this fact a theory concerning the nature of the process of the gray degeneration of the posterior columns in locomotor ataxy. In this case of secondary degeneration, the transparent material interposed between the tubes did not contain many nuclei, and did not offer a trace of amyloid bodies; peculiarities which connect this case with degenerations of long standing, such as we have described, and which separate it from the medullary sclerosis, such as we find in ataxy. During the same year, M. Cornil sent to the medical society of observation an example of compression of the cord, found in the service of M. Charcot. He says that he found the inferior segment of the cord normal. But an examination was only made of a few morsels of medullary tissue, and perhaps if he had made sections of the inferior portion of the organ, an abnormal production of connective tissue in the posterior portions of the lateral columns might have been recognized in

* Die graue degeneration der hinteren Rückenmarksstränge, p. 117; Berlin, 1863.

place of granular bodies which had disappeared. However, in another case, seen the next year by the same author, the descending degeneration, such as Türck had observed it, was indicated in the most exact manner. The compression, in this case, had been produced by a fracture of the vertebral column; and in the notes which M. Cornil was kind enough to give us, it is said that in the inferior segment the posterior columns were found perfectly healthy, but that the antero-lateral columns enclosed all through their substance numerous granular corpuscles, especially at the posterior part of the lateral columns.

About the same time, M. de Lacrousille showed to the anatomical society an epithelial tumor of the rachidian arachnoid which had compressed the cord and determined a paraplegia whose commencement dated back thirteen years. The patient had been observed in the service of M. Vulpian. In a report which I was deputed to make before the Anatomical Society upon this pre sentation, I pointed out the following results to which an examination of this cord conducted me. The compression which was situated above the lumbar enlargement had reduced the cord to such a degree that the meninges seemed to touch one another. Below the point compressed the cord was notably diminished in size, but the atrophy did not exclusively affect the antero-lateral columns. The examination was not made in the specimen in the fresh condition. In sections obtained after hardening it in chromic acid, a considerable rarefaction of the tubes was seen at the posterior part of the lateral columns, forming a transparent spot which in width came in contact with the meninges. This lesion diminished in extent as it went further from the point compressed, but it could be traced to the inferior part of the lumbar enlargement. The methods of pre-

paration employed did not allow of any search for the existence of granular bodies, which, considering the long duration of the lesion, had probably disappeared. As for the atrophy of the anterior columns and of the anterior portion of the lateral columns, it apparently resulted from the disappearance of a large number of nerve tubes, which disseminated through these fasciculi, had allowed the tissue to contract upon itself without leaving any empty space between the elements which remained.

The following year I had an opportunity of taking at the hospital Sainte-Eugénie, in the service of M. Tribolet, the observation of a little girl of thirteen years, affected with caries of the vertebræ at the lower part of the dorsal region. Death occurred about six weeks after the commencement of the paralytic symptoms which were the result of the compression of the cord by an intra-rachidian abscess. The portion situated below the point compressed showed a perfect integrity of the posterior columns, but numerous granular bodies were found through the substance of the antero-lateral columns, especially at the posterior portion of the lateral columns which, at the lower end of the organ appeared to be the only ones altered. No hypergenesis of the elements of connective tissue was found.

M. Charcot has communicated to me an observation of compression of the cord by caries of the vertebræ, taken in 1865, at the Salpêtrière, in which these descending lesions present the same characters. The paraplegia became complete only three days before death; nevertheless granular bodies were already to be found in the lower segment of the cord.

Finally, I have recently had an opportunity of observing at the Salpêtrière, in the service of M. Charcot, a case of compression of the cord produced by a cancer-

ous tumor developed in the posterior lamina of the first dorsal vertebra. Besides this, a collection of pus had softened in the rachidian canal and slightly compressed the cord on its posterior aspect, outside of the dura mater from the situation of the tumor to a point about one inch above the lumbar enlargement. The patient died five and a half months after the commencement of the paraplegia. All the columns were altered in the compressed portion; but below, in the lumbar enlargement, the posterior columns were healthy, while the lateral columns contained a considerable number of granular bodies, and a finely granular amorphous material, in which was found a large number of myélo cytes and other very elongated nuclei, resembling embryoplastic nuclei. In the anterior columns also granular bodies were found, relatively much less numerous; besides there were seen, between the nerve tubes, some myèlocytes and embryo-plastic nuclei as in the lateral columns, but in reality much less abundant.

From all these facts, it is clearly evident that, in cases of primitive lesion of the cord, the descending secondary degeneration occupies exclusively the antero-lateral columns; that the posterior columns always remain intact; that, in the antero-lateral columns, the principal and most extensive alteration is limited to the posterior portion of the lateral columns; and that the anterior columns and the anterior portions of the lateral columns also degenerate, but that, in these fasciculi, the degeneration rapidly diminishes, so as to disappear entirely at a short distance from the part originally injured.

Let us proceed now to the study of ascending degenerations of the cord consecutive to primary lesions of that organ. These degenerations have been observed eleven times by Türck. They implicate the posterior columns and the posterior portion of the lateral columns. The

anterior columns above the point primitively injured have always been found healthy.

The alteration of the posterior columns, which may occupy the entire surface of the section immediately above the lesion, gradually becomes narrower in proportion as it approaches the bulb, and leaves, at its external portion, the medullary tissue perfectly healthy. The granular bodies are become more and more limited to a zone which rests upon the posterior sulcus and upon the posterior surface of the cord. In the cervical region, they are only found in the smaller fasciculi, and the lesion disappears at the level of the floor of the fourth ventricle.

In the lateral columns, the granular bodies occupy the same situation as in the descending degenerations; but their number progressively diminishes as they approach the bulb. At this level, in place of affecting the anterior pyramid of the opposite side, as is observed in descending degeneration, they continue their course in the restiform body of the same side, pass upwards behind the olivary, and can once more be found at the insertion of the restiform body to the cerebellum, without having undergone any decussation in the substance of the medulla oblongata.

In the case of compression of the cord in a little girl, which I have reported above from Leyden, there was also an ascending degeneration, but this lesion implicated only the posterior columns; the nerve tubes were diminished in number and separated by a transparent material, just as in in the gray degeneration of the posterior cord in persons affected by ataxy. In the case presented by M. Cornil to the medical society of observation, it is said that they found above the point compressed some granular bodies in the posterior columns and in the neighboring portion of the lateral columns.

In the second case of M. Cornil, they also found that the posterior columns, above the lesion, presented a very large number of granular bodies, and they also found some of them in the antero-lateral columns, principally in the course of the vessels.

In the patient of M. Vulpian, who has been the subject of a communication to the Anatomical Society, I have found one of the most manifest ascending degenerations occupying exclusively the posterior columns. Numerous sections, made at different levels above the point compressed, gave me the following results: Immediately above the lesion, we find the tubes diminished in number throughout the whole substance of the posterior columns; a little higher up, we find upon each side a little band of healthy tissue applied upon the internal face of the posterior cornua; the alteration occupies all the remainder of the posterior columns, and presents altogether the figure of a trapezium, the two parallel bodies being formed, one in front, by the gray commissure, another, behind, by the meninges, the two others being parallel to the posterior cornua. In proportion as we depart from the primitive lesion, these two last borders approach one another more, at the same time that the little bands of healthy tissue, situated at the external parts of the posterior columns, increase in thickness. At length these two borders end by uniting in front at the junction of the commissure with the posterior sulcus. At this point, the altered portion appeared upon the section like the figure of an isosceles triangle, the base of which was behind on the meninges, the summit on the middle of the commissure, and the posterior sulcus formed the perpendicular dropped from the summit to the middle of the base. A little higher up, the lesion still preserved its triangular form, but it continued growing more contracted; the base which rested

upon the posterior part of the organ, diminished in length, and the summit left the commissure so as to approach little by little the base, following down the posterior sulcus; at length the alteration terminated at the level of the fourth ventricle. In this patient, the lateral columns presented no abnormal appearance.

In the little girl of thirteen years of whom I made an examination at the hospital Sainte-Eugénie, the granular bodies were seen only in the posterior columns above the tumor.

In the observation of recent compression by vertebral caries, which was communicated to me by M. Charcot, the granular bodies were also only found in the posterior columns above the point compressed.

It is not the same in the case of paraplegia from cancer of the vertebral column, recently observed by M. Charcot; the ascending degeneration affected at the same time the posterior columns and the posterior part of the lateral columns; it was characterized by the presence of granular bodies, of isolated fatty granulations, in large numbers and by the atheromatous appearance of the vessels in these parts.

We may conclude from an analysis of the preceding facts, that ascending degenerations of the cord are developed consecutive to primary injuries of that organ, and these secondary degenerations principally affect the posterior columns; that in these columns they gradually diminish in intensity, become limited little by little to the internal and posterior fasciculi, and terminate in a point at the floor of the fourth ventricle.

The alteration of the posterior part of the lateral columns is not mentioned in all the observations. It may be remarked, in this connection, that in the cases of Türck, in which this alteration was present, the

primary lesion was located quite high up the cord, at least above the middle of the dorsal region.

In one of the observations which I have taken, the cord was compressed just above the lumbar enlargement, and the lateral columns were intact. In another case this lesion was wanting, and the injury was located below the middle of the dorsal region. In the only case where I have found the lesion of the lateral columns, the compression was due to an alteration of the first dorsal vertebra. It would appear that the height of the primary lesion is not without influence on the alteration or integrity of the lateral cord from ascending degeneration. At all events this degeneration is of frequent occurrence and continues upward in the restiform bodies. Let us add in conclusion, that the anterior columns and the anterior part of the lateral columns are never altered by ascending degeneration.

VI. SECONDARY DEGENERATIONS CONSECUTIVE TO PRIMARY LESIONS OF THE POSTERIOR ROOTS.

Another ascending degeneration of the cord is observed as a result of primary lesions of the posterior roots; but we know of only one case which demonstrates this alteration: it was found in the ward of M. Trousseau, and the anatomical preparations have been presented to the society of biology by M. Cornil. An intra-rachidian tumor compressed the cauda equina without touching the cord; the nervous alteration implicated equally the anterior and the posterior roots; but as far as regards the secondary degenerations of the spinal cord, we may neglect the alteration of the anterior roots, which, according to the results of Waller's experience, degenerate only in the direction of the periphery. The posterior roots, on the contrary, injured above the ganglia which are annexed to them, should

degenerate in the centripetal direction. This is what was found in reality. Besides, the cord presented throughout its whole length a diminution of the tubes of the posterior columns. This rarefaction implicating the whole of the posterior columns at the level of the lumbar enlargement diminished gradually in intensity as it ascended, and became confined to the internal and posterior portions of the posterior fasciculi. However, the form of the alteration upon sections made at different levels was not identical with that which we have indicated for ascending degenerations from primary lesions of the cord itself. In place of being, as in the latter case, bounded laterally by two diverging straight lines, nearer one another in front than behind, the degeneration from injury of the cauda equina was circumscribed by the arc of a circle, the convexity of which was turned forward towards the commissure, and the two extremities of which rested upon the posterior face of the cord. The degeneration disappeared at the level of the floor of the fourth ventricle.

Before coming to the study of the functional troubles by which secondary degenerations of the cord betray themselves, we may deduce from the facts already demonstrated some conclusions relative both to the intimate nature of the process of these degenerations and their proximate causes, and to the normal structure of the spinal cord.

OF THE PROXIMATE CAUSE OF SECONDARY DEGENERATIONS.

We have already demonstrated that inflammation plays no part in the production of secondary degenerations of the white fibres of the cord; it only supervenes consecutively, if we may give the name of inflammation to that slow production of connective tissue which produces, so to speak, the cicatrization of the degenerated column. We have established the fact that the

first alteration affects the nerve tubes which undergo a granulo-fatty transformation analogous to that which has been observed in the peripheral portion of divided nerves. We have shown the analogy of this destruction of the nervous elements of the cord with that which is observed in cerebral softening from vascular obstruction, and we can now affirm the identity of the destructive process in secondary degenerations, and in degenerations of the nervous centres depending on arrest of the arterial circulation. In both cases there are found, after the first few days, granular bodies and a great abundance of fatty molecular granulations; the origin of these elements appears to be, in the softening as well as in the secondary degenerations, a destructive alteration of the nerve tubes. Some months since, I had an opportunity of observing with M. Charcot, in two cases of recent cerebral softening, the granulo-fatty infiltration of the cylinders of myeline of several tubes taken from the very seat of the softening, and presenting the same characters as those which I had been able to find at the commencement of secondary degenerations. But, when it is the result of a loss of the supply of blood, this destructive process seems to progress with more rapidity; it is for this reason that MM. Prevost and Cotard* have, in their experiments, been able to find numerous fatty granulations thirty-seven hours, and granular bodies three days, after the obliteration of a cerebral artery. They have also seen an atheromatous condition of the capillaries developed in points where they artificially produced a softening by cutting off the supply of blood. I have recognized in man, in a certain number of softenings from arterial obliteration, that this atheromatous condition of the capillaries is, as

* Société de biologie, janvier, 1866. (Gaz. Méd. de Paris, 1866, *passim.*)

in secondary degenerations, only an atheromatous appearance; the fatty granulations are, outside of the proper wall of the vessel, accumulated between that membrane and the lymphatic sheath. Finally, as the last analogy, let us state that, in softening from a defective supply of blood, as well as in secondary degenerations, a proliferation of connective tissue usually follows the destruction of the nervous elements, in that period of cicatrization of which M. Durand-Fardel has given so excellent a description.

Thus the same disturbance of nutrition producing a necrobiotic destruction of nerve tubes may be produced under the influence of two different causes, the loss of the supply of blood and the loss of nutritive action.

By the loss of nutritive action, we understand the cessation of the influence which the elements of the gray substance exercise on the nutrition of the nerve tubes, thus admitting the conclusions to which his experiments on the degenerations of the nerves have conducted A. Waller. We know that according to this physiologist, each tube of the peripheral nerves has at one of its extremities a nervous cell, which, independently of its special properties of innervation, has the function of presiding over the nutrition of the nerve tube which departs from it, and even of contributing by a peculiar influence to the reproduction of this tube, when it becomes degenerated at some point of its course.* These cells Waller has designated under the name of neurogénotrophes; we now call them more simply *cellules-trophiques*, (nutritive cells.)

* We should perhaps say with more precision at this time, that the centres exert a certain influence on the restoration and not on the regeneration of nerve tubes. It seems to result from the researches of M. Schiff, and of MM. Philipeaux and Vulpian, that the return of the functions of a divided nerve is produced, not by the production of new tubes in the midst of the débris of the de-

I cannot here refrain from examining an opinion which would tend to throw doubt upon the existence of these nutritive cells, or rather to deny the nutritive action which the nervous cells can exert upon the nerve tubes which arise from them. Under this hypothesis, the fatty degeneration of the nerve tubes could only be attributed to functional inertia. Reciprocally, if we admit that functional inactivity is capable of producing degeneration of nerve fibres, it is useless to accord to the central elements any influence whatever upon the nutrition of the tubes; this property would be superfluous, and for the cord, would cease to be demonstrable. Let us consider, then, whether the facts known up to this day permit us to admit that the nerve tubes can degenerate from the sole fact of functional inactivity.

If we cut a mixed nerve, the central extremity remains healthy; on the contrary the peripheral end degenerates, and the alteration implicates all the fibres, sensor and motor. If we consider only the motor fibres, the degeneration affects in truth only the trunk of those fibres, which no longer receives any impulse from the brain or the cord, which are consequently in a state of functional inactivity. It is the same when we divide the anterior roots; the extremity attached to the cord, and which receives from it motor excitation, remains healthy; the peripheral extremity, which no longer participates in this excitation, degenerates. Thus far nothing proves that the functional inactivity may not be the cause of the degeneration.

In the section of a mixed nerve, we have said, all the

generated tubes, as Waller thought, but rather by the reforming of the white substance of Schwann in the sheath of old tubes, around the axis-cylinder which, according to the same experiments, may remain for a long time, notwithstanding that it undergoes alterations which we are far from understanding.

fibres, and in particular the sensitive fibres, degenerate in the peripheral end; nevertheless, in this portion separated from the centre, they continue to undergo the excitations of contact, of temperature, &c.; they receive impressions which they cannot transmit to the centres; but the proper activity of the nerve tubes is put in play, notwithstanding which these tubes degenerate. On the contrary, the central end, which is certainly removed from all peripheral excitation, which is in the most complete functional inactivity, remains healthy. Now make a section in the middle of the posterior roots, and the degeneration affects the central extremity, the one which is attached to the cord, and which is in functional inactivity, and is entirely absent in the peripheral extremity which is in connection with the intervertebral ganglion, and which continues to receive external excitations. Thus, of the sensor fibres, the degeneration attacks sometimes those which are in functional inactivity, and sometimes those whose physiological activity continues to be provoked, and *vice versâ*. The functional inactivity then exercises no influence over the degeneration of sensor fibres. The degeneration affects only those portions which have lost their relations with the ganglia of the posterior roots, the central or medullary extremity of the posterior roots, and the peripheral ends of nerves.

The condition of integrity of a sensor fibre is therefore neither in the fibre itself, nor in its peripheral extremity which receives the excitations, nor in the cord: it is in the intervertebral ganglion, or, according to the opinion of M. Schiff, near this ganglion.

If the functional inactivity is without influence upon the degeneration or the integrity of the sensor fibres, we may, without pushing the analogy, admit that it is the same for the motor fibres. We then have the right

to state that the condition of integrity of a motor fibre is in the cord.

For ease of expression, we may translate these two propositions in the following manner: in nerves, the motor fibres have their nutritive cells in the cord, the sensor fibres have their nutritive cells in the ganglia of the posterior roots.*

If the integrity of the tubes of the peripheral nerves is due to the nutritive centres and not to the alternations of activity and repose, we are right in supposing that it is the same for the tubes of the cord, and that the degenerations which we have studied are due, not to the functional inactivity, but to the suppression of the action of the nutritive elements. Here experimental demonstration is impossible. In truth, in the cord, contrary to that which we have seen for the sensor fibres of the peripheral nerves, the degeneration of the tubes is produced always in the direction of their physiological activity. The antero-lateral columns, which conduct centrifugal excitations, degenerate below the point injured, that is to say, in the portion which is no longer excited; the posterior columns, whose conductibility is centripetal, alter above the point injured, in the portion which is thus reduced to functional inactivity.

But, to place a column of the cord in a state of inaction, it is not necessary to produce a lesion of the

* This word nutritive cells expresses more than our actual knowledge allows us to affirm, and perhaps it would be preferable to speak only of the nutritive elements, or still better of the nutritive centres. It is not demonstrated, in fact, that all the nerve tubes owe to their connection with a nerve cell all the nutritive activity necessary to the integrity of their structure. As regards the posterior roots, in particular, all of whose fibres have their nutritive centre in the intervertebral ganglia, it is well established that a certain number of these fibres only traverse the ganglion without contracting any relation with the bipolar cells.

column itself; we equally produce a functional inactivity of the fibres of the cord having a centripetal current, by destroying in the peripheral nerves the elements which connect mediately with them. This experiment is realized in amputations. Now, never, as a result of an amputation of the thigh, which nevertheless places in the most absolute inactivity all the fibres which were intended to transmit to the cord, and thence to the brain, the excitations coming from the lower limb, never, I say, has a degeneration been found of the posterior part of the lumbar enlargement, nor of any point of the posterior columns. This observation has already been made by L. Türck. In these cases, the fibres which remain inactive during long years preserve the integrity of their structure, because they have not lost their connection with their nutritive cells.

We can conclude then from all this discussion, that in the cord as in the nerves, the nerve tubes are placed under the dependence of the nutritive elements. The direction in which the degeneration will be produced, as a result of a lesion of a column, will indicate to us at which extremity of the tubes the nutritive elements are situated. We are now in a condition to draw from the facts just studied some conclusions relative to the normal structure of the spinal cord.

ANATOMICAL DEDUCTIONS.

In the secondary degenerations which follow primary lesions of the brain, of the cord, and of the spinal roots, the extent, form, situation and course of these degenerations indicate in the most exact manner the normal distribution of the nerve fibres which have been destroyed at the point primarily injured; and permit us also to study very exactly the intimate structure of the white columns of the spinal cord, their origins and their terminations, where the scalpel and the microscope

could only after great labor afford results less precise, often even very uncertain. L. Türck, first, sought to draw from the anatomo-pathological facts which he observed some deductions relative to normal anatomy; but he is rather anxious to deduce from it, by an interpretation which I do not think sufficiently strict, the direction of the physiological conductibility of different fasciculi of the cord. M. Gubler* also understood all the advantage that normal anatomy could derive from these facts, when he said in one of the conclusions of his memoir: "Thus the lines of softening in both directions (ascending and descending) studied by attentive observers, will serve to determine the situation and the arrangement of the sensor and motor fibres in the columns as well as in the nervous centres. Here pathology will still afford light to anatomy and to physiology." Besides, this method is not new: it is only the application to the cord of a process employed by Waller in the study of the peripheral nerves, and which he himself designated under the name of a "new method for the anatomical investigation of the nervous system." It is by this method that we have been able to study the distribution and anastomosing filaments of the nerves. I think I may say that there would be every advantage in artificially producing secondary degenerations of the spinal cord, so as to make an experimental application of the Wallerian method to the cord.

We have already seen that lesions of different parts, and even of parts very high up in the brain, determine secondary degenerations which can be traced throughout the whole length of the spinal cord: these degenerations also exist exclusively in the antero-lateral columns. We may therefore conclude that there are tubes in the whole

* Du ramollissement cérébral atrophique, (Arch. Gen. de Méd., 1859, t. 11, 6e conclusion.)

extent of these columns which have their nutritive cells in different parts of the brain. On the other hand, the descending degenerations diminish in intensity as they depart from the bulb; then the cerebral tubes which are prolonged into the cord have not all the same destination, but leave, in their course, the antero-lateral columns, so that only a very restricted number of them remain at the inferior portion of these columns. These fibres which abandon the antero-lateral columns do not escape by the roots; for in no case of secondary degeneration has an alteration of the spinal roots been noticed; we are then obliged to admit that they pass into and terminate in the gray substance.

The descending degenerations from lesions of the brain are not disseminated throughout the whole substance of the antero-lateral columns; they sometimes affect the internal portion of the anterior column on the same side as the primitive lesion, and this alteration of the anterior column disappears in the dorsal region; on the other hand, they always affect the posterior portion of the lateral column of the opposite side, and the degeneration continues as far as the inferior extremity of the cord. We conclude that the decussation of the pyramids is not complete; that some fibres, those of the external portion, which more rarely becomes degenerated, gain the internal portion of the anterior column, applying itself to the anterior sulcus, losing itself from point to point in the gray substance, and that the longest of these cerebral fibres do not go further than the middle of the dorsal region. This collection of fibres which the brain sends to the cord without decussation, and which is situated at the internal part of the anterior column, we will call the direct or internal cerebral fasciculus. As a second conclusion, we may state that the great majority of fibres which the brain sends to

the cord decussate below the pyramids so as to become located at the posterior part of the lateral column of the opposite side; that they preserve this position throughout the whole of their descending course; that they insensibly become lost in the gray substance, but that some of them are sufficiently long to attain the inferior extremity of the rachidian axis. This collection of fibres which pass from the brain to the lateral column of the opposite side, we will call the decussating or external cerebral fasciculus.

We have now built up only a small part of the antero-lateral columns; the study of degenerations consecutive to primary lesions of the cord will enable us to complete the description. We have stated that, when the cord is compressed at one point, the antero-lateral columns degenerate below the compressed portion, and that the alteration, though of but slight extent in the anterior columns, and in the anterior part of the lateral columns, is on the contrary very pronounced in the posterior and external portion of these latter fasciculi, and that in this situation it extends to the inferior extremity of the cord. Among the tubes which degenerate below the point compressed, there are some which come from the brain, and these are the ones of which we have spoken above. The direct cerebral fasciculi and the decussating cerebral fasciculi are in truth compressed at one point of their course, as is the remainder of the cord, and ought to degenerate in their inferior portion. It results from this, that at the internal part of the anterior columns, and at the posterior part of the lateral columns, the descending degeneration will implicate the fibres of encephalic origin. As it is only in this situation that there are any cerebral fibres, the degeneration of the remaining portion of the antero-lateral columns interests tubes of another class, tubes which do not come from the brain. The degener-

ation of these latter tubes follows a descending direction; they therefore have their nutrition cell above the point of compression. This nutritive centre is not in the brain, but it is above the point compressed; it is consequently in the gray substance of the cord above the point compressed.

There are, then, in the antero-lateral columns, independently of the fibres of encephalic origin, other descending fibres, medullary fibres proper, which arise from the gray substance of the cord itself. In order to understand the distribution of these latter fibres, we must recall, in an exact manner, the situation occupied in the cord by the fibres of encephalic origin. To render the description less obscure, I think I should insist more particularly upon the form and position of the decussating cerebral fasciculus. The study of secondary degenerations, consecutive to primary lesions of the brain, has shown us that it is almost cylindrical, and located in the substance of the lateral column at its posterior part, in front of the line of insertion of the posterior roots, in the angle formed by the meninges and the external face of the posterior cornu. It does not come in contact with the pia mater, but is separated from it by other white fibres, in such a manner that, when it has been destroyed by secondary degeneration, the sections of the cord show at the posterior part of the lateral column a hole, as if cut with a punch, in the healthy medullary substance, a narrow band of which separates it from the meninges. In secondary degenerations from lesions of the cord itself, this hole is larger; it has increased in front and on the outside, has come in contact with the pia mater, and is thus transformed into a depression. This is because the degeneration implicates not only the fibres of encephalic origin, but also the medullary fibres proper. And as the depression

which maps out the degeneration on sections of the cord, passes on towards the inferior extremity of the rachidian axis, gradually becoming narrower, we may conclude that certain medullary fibres descend, following the posterior part of the lateral columns, and become lost in the gray substance after going a long distance. We have already seen that these fibres have their superior extremity in the gray substance of the cord itself; they then establish relations between parts of the gray axis separated by quite long distances. We will designate these fibres by the name of long commissural fibres.

In compressions of the cord, the descending degeneration does not invade only the internal part of the anterior columns, and the posterior and external part of the lateral columns; the granular bodies are met with in the whole thickness of the antero-lateral column, but their number diminishes insensibly as we go further from the point compressed, and they disappear entirely at quite a short distance below the primary lesion. The fibres which thus degenerate through the whole substance of the antero-lateral columns, after a short descending course, are lost in the gray substance of the cord. They do not come from the brain, but they have their nutritive cell above the point of compression; they then arise from the gray substance of the cord, in which they also terminate at a short distance below their origin. I will denominate them the short commissural fibres.

Still this is not all: we have seen that in compressions which act upon the cord above the middle of the dorsal region a slight ascending degeneration of the posterior part of the lateral columns is observed; that this degeneration pursues its course in the restiform bodies, and even in the inferior peduncles of the cere-

bellum. We must therefore admit that this posterior part of the lateral columns, where we have already admitted the existence of two kinds of descending fibres, encephalic and medullary, contains also fibres of a third class, but in very small numbers: these should be fibres which have their nutritive cell at their inferior extremity. This nutritive cell we can only suppose to be in two points; either in the gray substance of the cord, or in the ganglia of the posterior roots. But we have seen that lesions of the posterior roots, which suppress the nutritive action of the ganglia for the cord, do not determine ascending degenerations of the lateral columns; we should then admit that the gray substance of the cord sends along the posterior part of the lateral columns some fibres which reach the cerebellum by the restiform bodies and the inferior cerebellar peduncles. In several cases of secondary degenerations, a vain search has been made for the trace of tubes which should go from the restiform bodies, across the deep portion of the pons, in the direction of the optic thalami.

In conclusion, the antero-lateral columns enclose encephalic fibres disposed in two fasciculi, at each extremity, at the internal part of the anterior column and at the posterior part of the lateral column. Besides, they are for the most part formed by descending medullary fibres proper, which establish relations between the different levels of the gray substance, commissural fibres, quite short in the anterior columns and in the anterior part of the lateral columns, much longer in the posterior part of these same columns. Finally, there exists also in this posterior portion of the white anterior substance of the cord, some ascending fibres which establish relations between the rachidian axis and the cerebellum.

L. Türck, to whom belongs the discovery of ascend-

nerations of the lateral columns, concluded from

this that these columns contained in their posterior portion, fibres with a centrifugal and fibres with a centripetal current. It is possible that the lateral columns may conduct centripetal impressions: certain physiological facts, upon which I may not insist, would tend indeed to make us think so. However this may be, this opinion cannot be legitimately deduced from the examination of secondary degenerations. We can affirm only one thing, which is that some of the fibres of the lateral columns have their nutritive centres at their superior, and some at their inferior, extremity. But the degeneration of a tube is not necessarily produced in the direction of its physiological conductibility: thus the sensor fibres of the peripheral nerves, the functional activity of which is exerted in the centripetal direction, have their nutritive elements at the superior extremity; so that the nutritive influence acts in an inverse direction to the physiological conductibility.

Let us now say a few words about the origin of the anterior roots, before passing to the study of the posterior columns. An experiment of Waller, already mentioned, proves that these roots have their nutritive centre in the cord. We have seen elsewhere that in compressions of the cord the anterior roots never degenerate either above or below the point primarily injured; from this we may conclude that they arise from the gray substance at a point very near their emergence, and that they run only a very short course along the antero-lateral column.

The constitution of the posterior columns seems to be less complex than that of the antero-lateral columns; but, in fact, their degenerations are not so completely known. From the special point of view which we take in this research, we can obtain our information from only two sources: from the study of compressions of the cord, and from that of the lesions of the roots.

Waller, in his experiments upon division of the posterior roots, had already remarked that, while the end attached to the ganglion remained healthy, the medullary end degenerated; and that "this disorganization could be traced for a short distance in the fibres of the posterior column of the cord, in the ascending direction."* Independently of these fibres, which are at once, or after a short course, lost in the gray substance, the posterior roots send others which pass through the columns for a much greater distance, and which are demonstrated in the case of compression of the cauda equina, which we have borrowed from M. Cornil. In this case, there was an ascending degeneration of the whole length of the posterior columns, and the alteration, very pronounced in the lumbar enlargement, gradually diminished in intensity so as to terminate in a narrow band, spread out under the meninges at the superior portion of the posterior pyramids. There are then some fibres which pass from the posterior roots of the lower part of the cord along the posterior columns, and are lost from point to point in the gray substance, some of them terminating only at the floor of the fourth ventricle. These fibres represent only a small part of those which the posterior roots bring to the cord; the remainder pass at once into the gray substance without aiding in the formation of the posterior columns. This fact is plainly established by microscopic anatomy and by physiology, but could not have been deduced from the study of secondary degenerations.

The ascending radical fibres which we have just pointed out, do not by themselves alone constitute the posterior columns. In fact, in this case of ascending degeneration from compression of the posterior roots, a

*A. Waller, Nouvelle méthode anatomique pour l'investigation du système nerveux: Bonn, 1852. Appendice: 8e conclusion.

section of the cord made at the lumbar enlargement, at a point where it had not yet received any healthy posterior root, showed, upon the section of the posterior column, quite a large number of tubes, disseminated through the midst of the sclerosed mass, which had taken the place of the radical fibres. These new tubes did not come from the roots; they did not have their nutritive cell at their superior extremity, since we know that compressions of the cord do not determine a descending degeneration in the posterior columns: we must then admit that they had their origin in the gray substance of the inferior part of the cord. The same may be said of other ascending fibres originating at different levels of the cord. We can demonstrate this proposition by comparing the form of the ascending degeneration in cases of compression of the roots, and in that of compression of the cord itself. When the degeneration is consecutive to a lesion of the roots, it is mapped out on the sections by a part of an ellipse, the convexity of the curve being in front; and, its two extremities resting on the posterior aspect of the cord, the tissue external to this line is perfectly healthy. When there is compression of the cord itself, these ascending radical fibres are injured at one point of their course and degenerate above the seat of compression; but the figure which the degeneration presents, upon sections of the cord, is no longer the same. In place of a segment of an ellipse, we have a triangle, the base of which is on the posterior face of the cord and the apex towards the commissure. This is because the degeneration also implicates other fibres which have their nutritive centre at their inferior extremity in the gray substance of the cord. These are the medullary fibres proper, like those which we have pointed out in the antero-lateral columns. In a case of compression of the

cord above the lumbar enlargement, I have seen the degeneration pass on retaining its same triangular form as far as the superior extremity of the posterior pyramids; the medullary fibres proper of the posterior columns therefore terminate in the gray substance after an ascending course of variable length. Some of them extend from the lumbar enlargement as far as the floor of the fourth ventricle. They have their origin and their termination in the gray substance; they therefore deserve the name of the posterior commissural fibres.

Ascending radical fibres and posterior commissural fibres are intimately mingled in the posterior columns without producing any confusion in their reciprocal distribution, since the form of the degeneration differs through the whole length of the cord, according as it implicates the former or the latter.

In the two cases of degeneration from compression of the roots or from lesion of the cord, the degenerated part, as it nears the bulb, becomes more and more limited to the posterior and internal portion of the posterior columns. We may therefore conclude that all the fibres of the posterior columns tend towards the posterior and internal portion of these columns, and are there located after they have run the greater part of their course. Then they probably curve forward and outward so as to terminate in the gray substance.

The distribution which I have just indicated is only exact for the fibres which come from the lower half of the cord. Of these fibres those which are prolonged as far as the superior part of the cervical region, are all situated in the substance of the small fasciculi and of the posterior pyramids. The fibres which originate in the upper half of the cord do not appear to mingle with the preceding, so that the sensor nerves of the lower limb and those of the upper limb should remain isolated

from one another, separated by the intermediate posterior furrows. Indeed, in a case of compression of the cord at the upper part of the dorsal region, L. Türck has seen the degeneration occupy the external portion of the posterior columns. Unfortunately, he did not make sections through the substance of the bulb nor that of the pons, so that the anatomo-pathological demonstration of the continuation of a part of the posterior columns through the restiform bodies, is completely wanting.

To recapitulate, the posterior columns are formed from fibres which come directly from the posterior roots, from commissural fibres, and probably also, at the upper part, from fibres which, following the lateral portions, reach the brain by the restiform bodies where they form relations with the ascending fibres of the lateral columns.

As a conclusion from all that precedes, we state, exclusively upon a basis of pathological anatomy, that we may consider the cord as essentially constituted by a gray axis, the different parts of which can doubtless communicate with each other even in the gray substance itself, but whose relations are also established through its whole length by commissural fibres, some anterior and others posterior. This gray axis should receive at its anterior part, and throughout its whole extent, fibres which come directly from the brain, it should receive at its posterior part and throughout its whole length, fibres which come from the ganglia of the posterior roots; these latter fibres being of two classes, some, direct, plunge immediately into the gray substance, the others ascending, only get there after a longer or shorter course. Then from the gray axis two classes of fibres should be given off; some should proceed towards the brain along the posterior part of the lateral columns,

and perhaps also along the external part of the posterior columns; the others should leave the cord at a point very near their origin, and should go to the periphery along the anterior roots.

Thus considered, the cord may be represented as formed of intrinsic parts, the gray axis and the anterior and posterior commissural fibres, and extrinsic parts, some afferent and others efferent. The extrinsic afferent parts should have two origins, one encephalic and the other peripheral. The extrinsic afferent parts, of encephalic origin, should only exist in the antero-lateral columns, and should establish relations of each of these columns with the two cerebral hemispheres; with the hemisphere of the same side by the direct or internal encephalic fasciculus, and with the hemisphere of the opposite side by the decussating or external encephalic fasciculus. The extrinsic afferent parts of peripheral origin should come from the spinal ganglia by the posterior roots, and should divide into two series of fibres, some plunging directly into the gray axis, the others also arriving at the gray substance after having assisted in the formation of the posterior columns. The extrinsic efferent parts should also have two destinations: some should pass up to the encephalic destination by the antero-lateral column, so as to reach the cerebellum; others, arising probably from the upper half of the gray axis, should reach the pons by the external part of the posterior columns. The others with a peripheral destination should emerge at once from the cord by the anterior roots so as to terminate in the muscles.

Thus the cord would form a necessary intermedial organ for all impressions which reach the brain from the periphery, and for all impulses which go to the periphery from the brain; no fibre would go directly from the brain to the muscles, or from the tegumentary surface to the brain.

This structure of the cord, based entirely upon pathological anatomy, is in perfect harmony with a large number of facts already established by the scalpel and by the microscope, and affords them complete confirmation; or rather, this harmony testifies in favor of the excellence of the method we have pursued.

The results at which we have arrived determine certain questions which were as yet doubtful; besides they seem to us to establish several new facts.

What we have said about the incomplete decussation of the pyramids has been known for a long time, and it was known in what parts of the antero-lateral column the fibres, which come from the pyramids, were located; but the terminations of these fibres were not so precisely determined.

The commissural fibres of the antero-lateral columns had already been admitted by Todd and Schroeder Vander Kolk; however we do not think that their distinction into two classes, according to their length and the special location of each of them, had been pointed out.

Notwithstanding the works of L. Türck, already published for some time, only very vague ideas were held about the ascending fibres of the lateral columns.

In conformity with the first opinion given by Schroeder Vander Kolk, we have shown that certain fibres of the posterior roots assist a great deal in the formation of the posterior columns; but we think that M. Dean has exaggerated their importance in considering the posterior columns as exclusively formed by these roots. We have, in fact, proved that there are in these columns a large number of commissural fibres admitted by Todd, by Gratiolet, and by several other anatomists.

As regards the fibres which pass from the cord to the brain along the posterior columns, pathological anatomy

has not yet given us sufficient information; however, we may say that, up to this day, other methods of investigation have not led to a greater degree of certainty: we are as yet reduced to physiological deductions.

SYMPTOMATOLOGY.

The clinical study of the secondary degenerations of the spinal cord has not yet been undertaken; and we may say that it was impossible to study them, when we only knew the first period of these alterations. When a nerve is destroyed at one point of its course, in such a manner as to suppress the relations of the centre with the periphery, the function of this nerve is abolished, and if it still possesses some activity of its own, it cannot display it by any manifestation either of sensation or of movement. We understand, from this, that the substance of the injured elements may become disintegrated without there being any new symptom in the paralyzed parts. We may then state, *a priori*, that the work of degeneration which is going on in a column of the cord does not actually betray itself by any symptomatic modification. Clinical observation fully justifies this presumption. In fact, while the lateral column of the paralyzed side commences to show manifest signs of degeneration by the sixth day, following the commencement of the apopleptic attack, we do not see the symptoms present any notable changes at that period; the primary contraction which is very rare in cerebral softening, and uncommon in hemorrhage, is usually earlier, and is observed from the commencement of the symptoms, or during the first few days. In every case, this symptom, which may exceptionally appear at the period when the secondary degeneration is produced, finds its explanation in the cerebral lesions which we may, with good reason, consider as complications, and

it should not be referred to the deuteropathic alteration of the spinal cord.

It is not the same for the contraction which comes on at a later period, a symptom almost necessarily consecutive to old cerebral lesions, and to which attention does not seem to us to have been sufficiently attracted. This contraction of the paralyzed parts which we almost invariably find in cases of hemiplegia of long duration, seems to us to have been wrongly referred to a chronic irritation of the brain, due to a contraction of the cicatrice of the primary seat of disease, or to the progressive march of an imaginary encephalitis. The cause of this permanent, tardy contraction seems to us to be in the cord. Certainly we cannot refer it to the granulo-fatty alteration of the tubes, an alteration which, as we have said above, cannot reveal itself by any symptom; besides, at the time when this contraction commences, the tubes injured in the brain are already destroyed throughout their whole extent. But the tubes of encephalic origin are mingled in the cord with other tubes which arise from the gray substance of the cord itself. These medullary tubes proper are then plunged into the midst of a tissue which, after a considerable period from the commencement of apopleptic symptoms, is the seat of quite an abundant proliferation of connective tissue. It is to the irritation of the medullary tubes by this neoplastic formation, it is to this secondary sclerosis, that we think we should refer the later contraction in cases of hemiplegia.

If this hypothesis is correct, we should meet with analogous symptoms in all those diseases, whatever be their nature and their location, which are accompanied by descending degeneration, with secondary production of connective tissue in the lateral columns; and these symptoms should closely resemble those produced by

primary sclerosis of these columns. We are thus led to seek what are, in these different pathological conditions, the common symptoms which can be referred to this common cause, primary or secondary sclerosis of the lateral columns.

It is principally in the modifications which supervene on the part of the motor apparatus that we shall find these symptoms common to the different diseases which are accompanied by secondary degeneration of the spinal cord. We must understand that these researches are not free from difficulties, not only on account of the complication of facts, which hinders their interpretation, but especially because much obscurity still reigns upon this point of the symptomatology of paralyses.

Of all the modifications which the condition of the muscles may present in cases of hemiplegia, the contraction is without contradiction the most important; it is also the one which has principally attracted the attention of observers. Early or tardy, temporary or permanent, it has always been regarded as an index of an irritation going on in the nervous centres. In accordance with Lallemand and MM. Cruveilhier, Bouilland, Andral, Durand, Fardel, Todd has especially insisted upon the semiological importance of this symptom, and has made it the basis of a division of cases of hemiplegia. He admits three classes of them, according to the conditions of the muscles. In the first, the paralyzed limbs remain flaccid and relaxed; in the second there is early rigidity of these muscles, and the rigidity makes its appearance at the very moment of the apopleptic attack, or a short time after. Finally, in the third category, the majority of hemiplegic cases enter, in which the paralyzed muscles, flaccid at the commencement, are attacked at a later period, and progressively, by a rigidity which becomes permanent.

This distinction which Todd has established and so strongly insisted upon, between the early rigidity and the tardy contraction is no less important in a pathogenetic than in a clinical point of view. "If the paralysis had been accompanied by rigidity, says the English author,* I should have been led to the conclusion that the cerebral lesion was of an irritating nature. This rigid state of the paralyzed limb (when it comes on at the same time as, or very soon after, the paralysis) is generally seen when some superficial part is affected, as the meninges or the surface of the brain, or when there is a growth from the skull, or a tumor in the hemispheres, or in some cases of inflammatory softening, or in some conditions keeping up a constant irritation; but when there is a simple rupture of the fibres of a deep-seated part of the brain, as the corpus striatum, with or without pressure, there is no irritation, and the paralyzed muscles are quite lax." And in another place:† "In the majority of cases the early rigidity is due to an apoplectic clot. My idea as to its cause is, that it depends upon a state of irritation, propagated from the torn brain to the point of implantation of the nerves of the affected muscles. But, you will ask, why is it that in some cases of clot, the hemiplegia will be accompanied with complete relaxation of muscles, while in other cases the rigidity of which I have spoken exists? The answer to this question is as follows: In the cases where there is no rigidity, the clot lies in the midst of softened brain, and has not in any degree encroached upon sound brain; but when rigidity exists, the clot has extended beyond the bounds of the

* Clinical lectures on paralysis, certain diseases of the brain, and other affections of the nervous system, by Robert Bentley Todd: lec. v. p. 100; London, 1856.

† R. B. Todd Lectures, 10 and 11 passim.

white softening, and has torn up, to a greater or less extent, sound brain. * * * This form of hemiplegia sometimes occurs in surgical practice, in consequence of a blow on the head, with depression of bone, or from considerable hemorrhage within the cranium, such as results from injury of the middle meningeal artery, or one or more of its branches. The paralysis of the opposite side is then accompanied by rigidity. * * * Sometimes inflammation of the pia mater, or arachnoid, causes an accumulation of fluid in the sub-arachnoid spaces, and then there is paralysis and rigidity. * * * An affection of the brain, of an irritating character, may give rise to this form of hemiplegia." All these assertions of Todd are based upon observations where autopsies were made; it is with justice that he states the encephalic seat and the irritating nature of the cause of early contractions in certain hemiplegias. Besides, this opinion does not materially differ from that of French pathologists. For a long time we have considered contractions in hemiplegia as a sign of encephalitis; and, besides this too absolute belief, a certain number of facts, several of which have been long known, have led us to admit that the hemorrhage determines immediate contractions, when the seat of disease, breaking up the convolutions, reaches the meninges, or when it bursts into the cavity of the ventricles. Then, is it not to an inflammation which is developed about the seat of disease, that we should refer the muscular rigidity which comes on a few days after the apoplectic attack?

According to this opinion, which nothing now seems to contradict, the muscular rigidity would result either from the irritation of the peripheral end of the fibres lacerated by the traumatic cause, or by the hemorrhage, or by the softening, or else from the irritation of the

fibres which surround the primary seat of disease, and which have preserved their integrity. According to the first hypothesis, as the lacerated fibres are destroyed after the sixth day, they should rapidly cease to be the seat of any phenomena of excitation. It would then be to these temporary contractions of the commencement that we could apply the opinion of M. Gubler* concerning the part of atrophic softening in the cessation of phenomena of excitation, and in particular of rigidity.

If we may, strictly, consider secondary degenerations as capable of putting an end to the muscular rigidity of the commencement, they play an entirely different part with reference to the later contractions; not, I repeat, that we can consider the contraction as the symptomatic expression of the work of granulo-fatty destruction of the tubes of the cord, nor even that we can attribute to the granules resulting from this destruction the power of irritating the neighboring tubes. The muscles remain flaccid and inert while this process of atrophy is going on; they only become rigid at a later period, when the hypergenesis of nucleated elements commences to appear in the degenerated column.

The later contractions of paralyzed muscles in cases of hemiplegia, are surely those whose clinical history is most imperfect; not that they have escaped notice, but their varieties, the date of their commencement, and the deformities (often characteristic) which they pro-

* Du Ramollissement cérébral atrophique (Archesis gén. de méd., 1859.) M. Gubler thus expresses the seventh conclusion of his Memoir: Clinical observation has as yet taught us nothing of the particular symptoms of secondary atrophic softenings; but we can forsee that after them we will observe a cessation of the phenomena of excitation, such as contraction, provided that the long duration of the primary affection has not given rise to such changes in the condition of the muscles as are opposed to the mobility of the parts.

duce, have as yet been only very incompletely pointed out. These contractions are, however, very frequent, and we may say that they are the rule in cases of hemiplegia of long duration. Out of thirty-two cases, the analyses of which will be given further on, I only once found the flaccid hemiplegia. I do not propose to trace out a complete study of these contractions: I will only endeavor to point out the most marked features of their history, taking as a basis the numerous facts which the clinique of the Salpêtrière can furnish.

When we examine a patient suffering from an old hemiplegia, we usually see the forearm of the paralyzed side is semi-flexed upon the arm, and we are assured that this position is invariably maintained against the influence of gravity by the tension of the muscles of the anterior part of the arm. The biceps in particular, even in cases of very long duration where it has undergone a notable atrophy, makes a more or less marked prominence, and it is evident to the touch that it is stretched like a cord between its insertions. If we tell the patient to extend the arm, he succeeds with great difficulty, even in those cases where voluntary motion is not entirely lost, in increasing by a few degrees the angle formed by the arm and the forearm, and then he is usually obliged to perform this movement mechanically, by pulling upon the paralyzed hand with the member of the healthy side. If the observer himself tries to produce this movement of extension at the elbow joint, he finds a variable resistance, which in some cases cannot be overcome. Usually, however, he succeeds in extending the forearm, at least within certain limits, and he experiences during this movement the same sensations as when the limbs of a dead body in the state of rigidity are made to move. The old cases of hemiplegia, in which the articulations of the paralyzed

arm are movable and loose, like those of a corpse when the rigidity has been overcome, are entirely exceptional. It is to these latter that we give the name of flaccid hemiplegias. We will regard all others as rigid, whatever may be the degree of resistance produced by the muscles, and whatever may be the number of muscles affected.

In hemiplegias of long duration, it is very rare to find all the muscles of one half the body paralyzed; it is equally rare to find voluntary motion injured to the same degree in all the affected muscles; it is also exceptional to find all the paralyzed muscles rigid. The contraction in cases of hemiplegia is never general; but it does not affect indifferently such and such a muscle, or such and such a group of muscles; on the contrary it presents a certain regularity in its determination.

I do not know that it has ever been observed in the muscles of the trunk, and it may be said to be limited to the muscles of the extremities. However, there is perhaps an exception to be made for the muscles of the face. Certain facts have led me to think that the muscles of the face, paralyzed at the time of the apoplectic attack, may afterwards undergo retraction, and produce a deviation of the features of the side of the face opposite to that where the deviation primarily existed. There are the cases in which the observations made at the commencement of the affection indicated a facial paralysis on the same side as the hemiplegia, with deviation of the features of the healthy side, and where, upon examining the patients some years later, I found that the features were, on the contrary, retracted on the same side as the paralysis, thus simulating an alternate paralysis.* As for the muscles of the orbit

* See Bouchard, Aphasie sans lésion de la troisième circonvolution frontale gauche, dans comptes-rendus de la Société de biologie,

and neck, the contraction of which so often turns the face and the eyes toward the side of the diseased hemisphere at the moment of the attack, we do not see that they are ever affected with a permanent contraction at a later period.*

Thus limited to the extremities, the muscular rigidity is not observed in the same degree in the superior and in the inferior.

It is always less marked and more limited in the pelvic than in the thoracic extremities; it may be absent in the former, while it exists to a more or less marked extent in the latter, while the reverse is not true.

Out of 31 cases of hemiplegia with rigidity, the contraction affected the muscles of the upper extremity 31 times, and the muscles of the lower extremity only 14 times. Even in the thoracic extremity, the rigidity is not always observed in all the muscles; and those which are rigid are not always so to the same extent. Thus we rarely see the shoulder elevated—10 times in 31 cases—and when it droops, which is more common—15 times in 31 cases—this result is due rather to the force of gravity than to muscular traction. We do not usually find any great difficulty in giving to the arm those different movements which the articulation of the shoulder permits; but, abduction is often quite limited. In its habitual attitude, the arm of hemiplegics is nearly always in a state of adduction, which the action of gravity is often sufficient to explain. Rarely, indeed, the arm is so strongly pressed against the thorax that

p. 111, année 1864. In this observation, there was no lesion of the pons capable of explaining the alternate hemiplegia, but there was a very pronounced secondary degeneration.

*M. Vulpian has insisted upon this symptom, which M. Cruveilhier had already noticed, and M. Prevost has made it the subject of an interesting memoir: De la Déviation conjuguée de la face et des yeux dans les hémiplégies. (Gazette hebdom., 1865.)

it may be necessary to attribute it to the contraction of the pectoralis magnus; however, this latter cause, though very evident in some cases, should usually be associated to a certain extent with the action of gravity; for, besides abduction being impeded, the humerus is frequently—18 times out of 31—in a more or less pronounced state of rotation inward.*

While the muscles of the regions of which I have just mentioned are contracted in only a slight degree, or even not at all, the muscles of the arm, of the forearm and of the hand, are always completely or partially contracted, and, in every case, it is in those parts that the tardy rigidity is the most pronounced.

The contracted muscles, triumphing over those which are only paralyzed, produce special and permanent attitudes of the limbs; but those remarkable deformities which we find in old cases of hemiplegia, result not only from the predominant action of the paralyzed and contracted muscles over those which are attacked by flaccid paralysis; more frequently, indeed, the deformities are the result of the contraction of antagonistic groups of muscles. This is easily demonstrated for the muscles of the arm and forearm.

The joints which the contraction has placed in a fixed position communicate to the observer a notable resistance, whatever be the direction in which he tries to move them. The elbow, for example, which is usually semi-flexed in cases of hemiplegia, is sometimes as difficult to put in a condition of complete flexion as in that of extension.

Often, however, the forearm presents in both directions a very limited degree of mobility, compatible with

* We shall see that, out of 31 cases of rigid hemiplegia, I have 12 times seen the arm in adduction, once in abduction, and 19 times in an indifferent position.

some very restricted voluntary or communicated movements; but if we try to increase the movement, we suddenly experience the resistance which I have mentioned above. These movements which we endeavor to produce are alike painful in flexion and extension. Finally we can determine by the touch that the antagonistic muscles are equally contracted. While the biceps is stretched like a cord, we feel the triceps hard and rigid.

We might suppose that this contraction of antagonistic groups of muscles was only a spasm, a reflex convulsion provoked by the pain induced by the motions which we give to the paralyzed limbs, something similar to the muscular retractions in arthritis or arthralgia. But in these cases the inhalation of chloroform suddenly puts an end to the spasm and restores their entire freedom of motion to the joints; while I have convinced myself, with M. Charcot, that this is not the case with rigid hemiplegias. Several patients having been put under the influence of chloroform, we have seen that while all the other muscles became relaxed, the contracted muscles preserved a marked rigidity, to a less degree, however, than when the patients were not under anæsthetic influence, but sufficient to prevent complete extension or flexion, and to reduce to their primary position the limbs whose attitudes had been modified. If we continued the action of chloroform for a longer time, we then saw one of the antagonistic groups yield a little to the more powerful traction of the opposing muscles, and produce a slight change in the position of the articulation; then this change once produced remained, even after the patient came out from under the influence of the anæsthetic, and the limb gradually returned to its former position, but not until several hours after the cessation of the inhalations.

The permanent vicious attitudes in hemiplegias of long duration are then for the most part the result of the opposing action of antagonistic groups of muscles contracted in a relatively greater or less degree. Hence it is that the position of the limb is generally intermediate between those which would be produced by the isolated action of these groups of muscles, more or less flexed or more or less extended, according as the contraction is stronger in the flexors or extensors. This fact is made evident by a symptom which we can produce at will in certain hemiplegics, and particularly in those having a unilateral atrophy of the brain resulting from an affection of that organ, dating back from infancy. The usual attitude of these patients consists in a flexion of the forearm with pronation and flexion of the hand and fingers. If by force we extend the fingers, we see that at a certain point they come spontaneously and suddenly as the movement of a spring into a condition of forced extension, at the same time that the flexion increases in the radio-corpal articulation, and the limb remains in this new posture for an indefinite period. If we then induce flexion, we at first experience a certain amount of resistance, then suddenly again the flexion of the fingers is spontaneously completed, and the hand is slightly straightened; thus the primitive attitude is reproduced. Out of 14 patients in the service of M. Delasiauve, suffering from cerebral atrophy, I have observed this symptom twice, and I have found it once with M. Charcot in one of his patients who had cerebral softening dating back thirteen years.

The vicious attitudes produced by permanent contraction in hemiplegics are very variable; they are principally remarkable in the upper extremity. If we consult the table in which are noted the 31 cases of rigid hemiplegia upon which this description is based,

we see, as I have said above, that the shoulder is elevated 15 times, lowered 10 times, in an indifferent position 6 times; that the arm is adducted 12 times, abducted once, in an indifferent position 18 times; that it is rotated inward 18 times, and that 13 times it does not show any tendency to rotation in either direction. We see, besides, that the elbow is flexed 27 times, extended 3 times, in an indifferent position once; that the forearm is pronated 24 times, supinated 7 times; that the hand is flexed 16 times, extended 15 times, in an indifferent position 10 times; finally that the fingers are flexed 28 times, extended 3 times.

The hand being always in a determined position of pronation or of supination, and the fingers being always either permanently flexed or extended, we may arrive at the following conclusion: that of all the muscles of the economy, those of the forearm are most often generally affected with permanent contraction in cases of hemiplegia; hence the precept that we should examine, in cases of softening or hemorrhage of the brain, dating back for some time, whether the movements of rotation which we give to the radius are equally easy in both directions, and whether the fingers can be completely straightened or flexed without difficulty.

Many of these partial attitudes which I have just pointed out are habitually grouped together and produce deformities of the whole region, the various elements of which do not appear related to one another according to invariable rules, but in which it is nevertheless possible, amid very numerous exceptions, to recognize certain general types. In order to characterize these types, we should take as a starting point the partial deformities, which are never absent, that is to say the condition of pronation or supination of the forearm, and the state of flexion or extension of the fingers. We

may thus artificially create four varieties, which, according to our observations, are distributed in the following manner: out of 31 cases of rigid hemiplegia, we find 22 times pronation with flexion of the fingers, twice pronation with extension of the fingers, 6 times supination with flexion of the fingers, once supination with extension of the fingers. Adding now the condition of extension or of flexion of the articulation of the elbow, we should double the number of these varieties, but clinically we have only been able to find the six following varieties:

Flexion of the elbow,			pronation and flexion of the fingers,	18 times.
"	"	"	pronation and exten. of the fingers,	2 "
"	"	"	supination and flexion of the fingers,	6 "
"	"	"	supination and exten. of the fingers,	1 "
Extension of the elbow,			pronation and exten. of the fingers,	3 "
Indifferent position of the elbow,			pronation and flexion of the fingers,	1 "

An examination of this table shows that there is no need of thus multiplying the varieties, and that for the clinique we should recognize one great type, the type of flexion characterized by the simultaneous flexion of the elbow and the fingers, or, in the absence of rigidity in the elbow joint, by the simple flexion of the fingers. We will also place in this type of flexion, the cases in which the fingers being extended the flexion of the elbow is so pronounced, that the angle formed by the arm and the forearm is less than 135 degrees.

Another type much less frequent will be characterized by the complete extension of the elbow, whatever be the condition of the fingers, or by the extension of the fingers, provided that the angle of flexion of the elbow be more than 135 degrees.

By consulting the table in which the angles of flexion of the elbow are indicated, we will see that the type of

flexion is observed 26 times, while we only meet with the type of extension 5 times.

The condition of pronation or of supination of the forearm adds two varieties for each of these types: thus clinically, we may admit four forms of deviation of the upper extremity in hemiplegias with tardy contraction, and these forms are observed in the following order of frequency:

Flexion with pronation,	20 times.
" " supination,	6 "
Extension with pronation,	4 "
" " supination,	1 "

We will describe only the first form, which is by far the most frequent. In this form we have seen the shoulder lowered 9 times and elevated 7 times; in four cases only, was the shoulder of the diseased side on the same horizontal line as the other. The arm is usually drawn to the body, either by its weight, or by a slight contraction of the *pectoralis magnus;* it is in this form that we have met the only case of abduction of the arm which we have observed. In the 20 cases in which there was flexion and pronation, we have 11 times seen the hand flexed with the fingers; once the hand flexed while the fingers were extended; once the hand extended while the fingers were flexed; finally, the fingers were flexed 7 times while the hand was in an indifferent position. In the case where the flexion of the elbow was most pronounced, the angle formed by the forearm with the arm was 30 degrees.

In this form of flexion with pronation which we usually meet in hemiplegics, the arm is drawn toward the trunk, and, owing to the rotation of the humerus, the forearm is applied against the body; the hand, usually flexed as well as the fingers, is, according to the degree of flexion of the elbow, pressed against the ab-

domen or against the thorax, and the parts in contact vary according to the degree of pronation of the forearm. In a first degree, the hand is in contact with the trunk by its palmar surface; in the second degree, by its radial edge; in the third, by its dorsal aspect; in the latter case, the elbow is more or less carried forward.

In the type of extension, we can still find these three degrees of pronation. The third was not shown by any of the patients referred to in the table, but it was very marked in the case of a woman in the service of M. Charcot, who has recently died from an old softening. In this woman, the left forearm was completely extended, the hand flexed at a right angle, and the fingers firmly folded up in the palm. The movement of rotation had carried the hand directly outward, the ulnar edge was in front, the radial behind, and this deformity had been increased by a rotation of the arm which had brought the olecranon directly in front. In the case of this woman, whose medulla oblongata I have shown to the Anatomical Society, the descending degeneration had advanced to an extent which we rarely find: the secondary sclerosis of the cord was visible as far as the inferior extremity of the left lateral column, and I should add that the sclerosis reached the meninges towards the middle of the dorsal region, instead of forming a little band, completely surrounded by healthy white substance. I was then too absolute in stating in a former part of this work, that no fibre of the decussating encephalic fasciculus came in contact with the pia mater. In this woman, besides, the flexion of the fingers was such, that the nails in growing cut the skin of the palmer surface. This occurrence, which is not very rare, and which has already been noticed by Todd,*

*Loc. cit., lecture x.

produces very painful ulcerations which secrete an infectious discharge; great care should therefore be taken of the hands and nails; it is prevented by permanently placing in the palm of the hand a roll of bandage, which is sufficiently held there by the contraction of the fingers.

I should say in conclusion of what has reference to the deformities of the hand, that when the hemiplegia commences, before the complete development of the individual, and above all in infancy, the hand which is usually flexed, instead of showing the flexed joints by sharp angles, presents on the contrary upon its dorsal portion a regularly convex surface which continues without interruption from the forearm to the last phalanges. This peculiar form is doubtless the result of an atrophy of the osseous tissue and of the articular prominences; an atrophy in which the subcutaneous cellular tissue does not participate. This character is sometimes sufficient, in the absence of any history, to distinguish a former softening in the adult from unilateral cerebral atrophy consecutive to some lesion which has destroyed a more or less considerable portion of one hemisphere during infancy.*

I shall pass more rapidly over the tardy contraction of the lower extremity in hemiplegics, and the deformities which are its consequences. I shall only state that out of 32 cases of hemiplegia of long duration I have found muscular rigidity in the pelvic extremity only 14 times; the hip was rigid 10 times, 4 times flexed, 6 times extended. The flexion was 3 times accompanied by abduction; the extension was 4 times accompanied by abduction. The knee was found flexed 10 times;

* Consult upon this subject a memoir presented by M. Cotard to the Society of Biology in 1865: Note sur quelques cas d'atrophie cérébrale; de l'attitude des membres paralysés dans cette affection.

22 times the limb was in the axis of the thigh without any appreciable rigidity. These 10 cases of flexion of the knee coincided 5 times with extension of the hip, 4 times with flexion and once with relaxation of the coxo-femoral articulation. The foot was found rigid and out of its proper position 11 times, 9 times presenting the type of talipes equinus and twice that of talipes talus. Of the 9 cases of talipes equinus, 5 were accompanied by flexion of the knee. The 2 talipes talus coincided with a considerable degree of flexion in the femoro-tibial articulation.

At what period and in what manner is this tardy contraction developed in hemiplegics?

This is a question which has scarcely been proposed, and which is far from having been decided. Todd, who has distinguished so carefully between the precocious and the tardy rigidity, says that he has met with the latter one year after the apoplectic attack, and he quotes from M. Andral a case where the contraction came on three months after the commencement of the hemiplegia. We may see that in one patient, whose case is referred to in the table, the flexor muscles of the fingers were rigid at the end of four months, but in her case the rigidity had already existed for some time. We have had an opportunity, in the case of this woman, to follow the development of the disease from its commencement, and her case is interesting in more than one respect. She was suddenly attacked at the age of 66 years, with apoplexy with complete loss of consciousness and left hemiplegia, without having presented any prodromic symptoms. The following day the intelligence had entirely returned, the paralyzed muscles were flaccid and there was no fever. The temperature of the rectum varied from morning to evening between 100° and 98.9°. At the end of eight days the fever came

on; the temperature went up to 101.1°; there was excitement with delirium, and the paralyzed muscles became rigid. Two days after, the temperature came down to 99.6°; the intelligence was restored and the muscles had become flaccid again. From this time the general symptoms disappeared, but the paralysis remained. Three weeks after the commencement of the illness the fingers were semi-flexed, but could be easily extended: however, forcible extension appeared a little painful. Two months after the attack the flexion of the fingers was more pronounced, and quite a marked resistance was experienced when we tried to extend them, an operation which seemed to produce considerable pain; the forearm was slightly pronated, and resisted slightly any movements of supination which we communicated to it; it presented a very slight amount of flexion; we were able to increase this flexion without hurting the patient, but when we afterwards extended it, we observed that after having easily reduced it to its former position we experienced a sudden resistance, of such a kind that the extension could only be completed by an effort which was painful to the patient. At this time there were no cerebral symptoms and no fever.

In this case we see a precocious contraction connected, as the delirium and fever indicate, with slight symptoms of secondary encephalitis; then we see a contraction become insensibly developed, traces of which we can barely find at the end of three weeks, and which is no longer doubtful at the end of three months. It is this latter contraction, which has since increased while the cerebral functions have returned more and more to their normal condition, that we think should be referred to the sclerosis secondarily developed in the place of the degenerated fibres of the lateral column.

This case also shows that if the muscles of the fore-

arm are the ones usually affected by tardy contraction, it commences likewise in them. This contraction comes on gradually and insensibly; this is the reason why we can hardly ever obtain from the patients information of any value concerning the time of the appearance of this symptom. They know that at first their limb did not offer any resistance to the movements communicated to it, and that at a later period it was fixed in one permanent attitude which could only be altered by a certain degree of force, but the transition between these two conditions has been so gradual that usually it is impossible for them to tell, even approximatively, the time of the commencement of the contraction. The determination of this time is almost as difficult for the physician who is watching the development of the disease; so that he had better not give the result of his observations except after quite long intervals, the changes undergone by the muscles from day to day being entirely inappreciable. In the preceding case, the contraction was evident at the end of two months, but it already existed before this time, and I may state that, having inquired into this question of a certain number of patients, I think I may conclude from their replies that while the limb was flaccid during the first month, it already presented a vicious attitude during the third month. It would then appear that the permanent contraction habitually commences in the course of the second month. It is plain that new investigations are necessary to determine this point, as yet obscure, in the symptomatology of hemiplegias.

The contraction commences in the muscles of the forearm; generally the fingers are flexed and the forearm is pronated; then, in most cases, the elbow becomes flexed, and, while the fingers curve more and more, the flexion of the elbow progressively increases in such a manner,

that for a long time the attitude of the limb is changed, showing it more every day.

I have remarked that in the patient of whom I have just spoken the straightening of the contracted parts was painful from the commencement, even when a slight effort sufficed to overcome the resistance of the muscles, and then these explorations resulted in momentarily increasing the rigidity. On the contrary, when the contraction is final, the muscles which we extend by force oppose during a considerable time the movements which we communicate to the limbs.

Sometimes, upon lifting the contracted arm of a hemiplegic by the end of the fingers, we see the entire limb agitated by a rapid trembling similar to that which we produce by the same proceeding in the inferior extremities of patients suffering from compression of the cord. I have only met with this symptom in the thoracic extremity in hemiplegia, and I think that we may consider it as an exception.

When the contraction has reached its complete development, it may notwithstanding present momentary variations in its intensity under the influence of certain circumstances, such as emotions or pains along the course of the nerves of the limb; in young women having hemiplegia, we sometimes see the contraction increase during the menstrual period.

I have already spoken of the action of chloroform upon contracted muscles; I shall also say some words of electricity. The induced currents which usually produce in the flaccid muscles of hemiplegias of short duration, slighter contractions than in the normal condition, produce on the contrary a marked and sometimes an exaggerated effect upon the muscles affected with permanent rigidity, even when they have undergone a certain amount of atrophy. But when this

atrophy affects unequally the different muscles of a limb, we can see, as I have found in one case, that by applying the poles to the muscles which act against the deviation, this deviation, far from diminishing, on the contrary increases. In this case, no doubt, the currents traverse the atrophied muscles and influence their antagonists so as to increase their already predominant power.

Once established, the tardy contraction may last for long years, often during the whole life of the individual, but sometimes it seems to diminish. Then however, the muscles cannot undergo a sufficient amount of extension, and the articulations present certain alterations which hinder any complete straightening, and the vicious attitudes remain; they are then passive. One might also ask whether we can hope for a cure of the hemiplegia when the muscles are already affected by tardy contraction. I do not know any fact which can encourage this hope, and the patients sometimes are deluded with reference to this subject.

Voluntary motion is not always totally abolished in the paralyzed limbs of hemiplegics, but the contraction limits and renders more difficult the muscular actions which are still under the influence of the will. If the rigidity diminishes, then these movements recover more liberty, and such a patient who is always to remain impotent imagines that he sees in this modification the commencement of his cure.

Some symptoms which have a very great analogy to these which we have just studied are observed in certain diseases of the cord which are accompanied by secondary sclerosis of the lateral columns, and especially in cases of compression of that organ. During the first period which correspond to the granulo-fatty degeneration of the tubes, the paralyzed muscles remain flaccid;

then, at a more or less advanced period, the contraction appears, the muscles atrophy and the lower limbs assume permanent attitudes which it is difficult to overcome. In some cases, however, the muscles remain flaccid and the limbs are swollen with an elephantiasic œdema; this is on account of the softening of the inferior portion of the cord. The phenomena of excitability, on the contrary, are observed in those cases where the autopsy reveals a secondary descending sclerosis. I need not insist in detail upon these symptoms, which have been very well described, especially by MM. Louis, Cruveilhier, and Brown-Séquard,* nor upon the characters of the contraction, for it is in every respect identical with that of patients suffering from hemiplegia. I think that, in order to explain these facts, it is not necessary to consider that there is an accumulation of nervous influx in the inferior part of the cord; we have here an irritation of this inferior extremity, as M. Brown-Séquard thinks; but this irritation has its anatomical cause.

A woman in the wards of M. Charcot, suffering from an ulcerated cancer of the breast, the commencement of which dated back for six years, was suddenly seized with violent, lancinating pains in the lower extremities; for two months, however, the patient had enough strength to be able to walk with the assistance of a cane. At the end of this time, she became unable to stand and was obliged to keep her bed; her legs were flaccid and inert. About seventy days after the time when she became bedridden, her limbs commenced to become flexed, and this flexion, slightly pronounced at first,

* Louis, Recherches d'Anatomie Patholog. Cruveilheir, Anatomie Patholog. du corps humain. Brown-Séquard, Lectures on the diagnosis and treatment of the principal forms of paralysis of the lower extremities: London, 1861.

progressively increased. The legs were strongly flexed upon the thighs, the thighs were in a state of adduction with a certain amount of flexion. A considerable amount of resistance was experienced when we endeavored to extend the contracted limbs, and these attempts were very painful to the patient. The sensibility was preserved; there was a certain degree of hyperæsthesia and some pains in the lumbar regions, with a very painful feeling of constriction around the abdomen and at the base of the lungs. Although purulent during the last few days of life the urine remained acid until death. The patient died the eighteenth of January, 1866, about six months after the commencement of the paralytic symptoms. The autopsy showed that the cord was compressed by a cancerous tumor of the first dorsal vertebra and by a purulent effusion in the canal, extending from the seventh cervical to the tenth dorsal vertebra. Independently of the ascending degeneration, in the cervical region, we found throughout the whole substance of the lateral columns of the lumbar enlargement, a considerable number of myelocytes and embryoplastic nuclei. The muscles of the posterior portion of the thigh showed no transverse striæ, either by direct or oblique light; the primitive fibres were studded with numerous fatty granules, (resisting the action of acetic acid;) the nuclei of the sarcolemma were extremely numerous.

In this case also, the contraction commenced about two months after the début of the paralysis, precisely at the period when the proliferation of connective tissue becomes developed in the secondarily degenerated columns. In another case of compression of the cord, by Pott's disease, which I examined at the hospital Sainte-Eugénie, with M. Triboulet, death having taken place one month and a half after the commencement of the

paralysis, I noticed that the muscles were flaccid all the time, and at the autopsy I found only a fatty degeneration of the lateral columns below the seat of the lesion without any hypergenesis of the elements of connective tissue.

We see that the permanent contraction which is common to hemiplegias and to compressions of the cord, presents in both cases the greatest analogy as regards symptomatology; it is connected with an anatomical condition of the cord common to both these affections, and does not become developed in either case until the time when the secondary sclerosis of the lateral columns commences. I should add that the permanent contraction of the extremities is seen to have the same characters in primary sclerosis of the lateral columns, as is shown by a remarkable case communicated by M. Charcot* to the Medical Society of the hospitals, as well as in certain cases of diffused sclerosis of the cord, affecting in a greater or less degree these same lateral columns, examples of which are to be found in a recent work presented by M. Vulpian,† to the same society. Why then, since in all these cases we observe common symptoms and common lesions, do we refer the contraction, in paraplegias, to the lesion of the cord; in hemiplegias, to a lesion of the brain?

This cerebral origin of the tardy contraction is not proved; it was admitted at a time when we were ignorant of the secondary alterations of the cord, and when we considered softening as a chronic encephalitis. It is based therefore upon a double error.

If we admit that the tardy contraction in hemiplegia results from a secondary sclerosis of the lateral columns,

* Hystérie avec contracture sclérose des cordes latéraux, 1864.

† Note sur les scléroses en plaque de la moelle épiniére, 1866.

we shall have a reason for certain facts which would otherwise appear inexplicable. Thus, in compressions of the cord, the contraction of the inferior extremities is stronger than in hemiplegias, because in the hemiplegia the secondary sclerosis is only developed in the situation of one portion of the decussating encephalic fasciculus, while in the compression of the cord it occupies not only this entire fasciculus, but a certain number of long commissural fibres besides. Thus, also, in hemiplegias, the superior extremity is most often contracted, and is so to a greater degree than the lower extremity, because the decussating encephalic fasciculus is richer in nervous fibres in the cervical region than in the lumbar region, where it terminates in a point; the sclerosis, which is substituted for this fasciculus throughout its whole length, will therefore be more developed at the level of the origin of the nerves of the arm than at the point of departure of the nerves of the pelvic extremity. Thus, also, may we not say that if the thoracic extremity is strongly contracted, while the head is not perceptibly deviated, that it is because the rotator muscles of the head are partly supplied with nerves from the spinal accessory which arises from the lateral portions of the bulb and from the superior extremity of the cord at points which are not influenced by the secondary sclerosis, since, in this region, it is limited to the internal part of the anterior pyramids? But it might be objected that all hemiplegias are not accompanied by contraction. We can answer this objection by stating that there are certain cerebral lesions, as for example superficial lesions of the convolutions, which are capable of producing hemiplegia, and which do not determine secondary degeneration.

We might refer the tardy contraction to other causes, such as an alteration in the structure of the

muscles due to the prolonged inertia, but we find complete hemiplegias which always remain flaccid; or to an atrophy of the muscles, resulting either from rest or from some other cause, but the contraction is found in 9 out of 31 cases, without there having been any atrophy of the muscles, and in one case I have found a certain amount of atrophy of the muscles of the arm, without any contraction. There are in reality alterations in the structure of contracted muscles coinciding with a fawn-colored appearance of their tissue; the transverse striæ are often less marked than usual, sometimes absent, the substance of the primitive fasciculi is more or less granular, studded with fatty and pigmentary granules; the nuclei of the sarcolemma increase in number; 16 times out of 30 the size of the muscles diminishes.* If these alterations are the proximate cause of the contraction, of which we have no proof, nothing authorizes us to consider them as developed without the action of the central nervous system.†

Independently of the contractions, one quite important symptom seems to me to be attributable to de-

* Out of 30 cases of hemiplegia with contraction, I have 5 times observed an increase in size of the paralyzed limb, but this hypertrophy no doubt depended upon the œdema of the subcutaneous tissue rather than upon an alteration of the muscles.

† M. Charcot has called my attention to a very curious peculiarity of paralyzed and contracted muscles in hemiplegias of long duration: this is the absence of post mortem rigidity. At the autopsy the limbs of the healthy side present a perfect rigidity; on the contrary the muscles which were contracted during life are entirely flaccid. However, numerous examinations made at different hours after death have shown that usually the diseased muscles do not escape the post mortem rigidity, which is manifest in them almost immediately after death, and only for a very short time. The absence of cadaveric rigidity is seen also in infantile paralysis. It would be curious to find out whether putrefaction is developed more rapidly in the paralyzed limbs.

scending degenerations, and more especially to the secondary sclerosis of the bulb. I refer to the epileptiform attacks and to those which are evidently epileptic, which we often meet with in subjects attacked with hemiplegia during infancy, and which are not infrequent in old persons suffering from softening, and in which we find at the autopsy considerable atrophy of a peduncle, of the pons and of the bulb. I must confess that this hypothesis does not as yet appear to me susceptible of a rigorous demonstration; but it seems to me to be true, because in one patient suffering from an intense sclerosis of the bulb from compression of that organ, the epileptic fits were very strong and very frequent, and because when we saw him sometimes, a few moments previous to an attack, the contraction markedly increased in the paralyzed limb.

We very often see in cases of hemiplegia an increase in the size of the nerves, with vascularity and increased thickness of the envelope of connective tissue, often also with a deposit of fatty globules in its interstices. I do not know whether this kind of hypertrophous neuritis, to which M. Charcot, and after him M. Cornil,* have called attention, depends upon secondary degeneration of the cord, or whether it is not solely the result of inertia; at all events, it seems to be connected with those pains, often quite severe, already pointed out by Remak, which the hemiplegics feel in the paralyzed arm, pains which are increased by pressure along the course of the nerve, and which are often alleviated by the application of a blister, as M. Charcot has several times proved.

As for the alterations of nutrition, such as the atro-

*Note sur les lesions des nerfs et des muscles liées à la contracture tardive et permanente des membres dans les hémiplegies. (Comptes-rendus de la Société de Biologie, 1863.)

phy of the compact tissue of bone, the squamous condition of the skin, &c., I doubt whether they can be referred to the secondary degeneration of the cord.* This degeneration, besides, does not modify in any respect the phenomena of calorification which we observe on the side of the paralyzed limbs. The paralyzed hand is always the warmer, even at a time very distant from the commencement of the symptoms, and we sometimes find considerable variations in the temperature of the two sides of the body.†

The examination of five patients, made with this object in view, has furnished me with the following results:

Age.	Date of commencement.	Paralyzed hand.	Healthy hand.
70 years.	Several years.	95.3°	89.6°
42 "	14 months.	95°	89.9°
72 "	12 years.	99.3°	98.6°
65 "	5 "	97.8°	97.5°
51 "	14 months.	97.1°	95°

Thus far I have only spoken of the symptoms of descending degeneration; the secondary ascending scleroses do not appear to betray themselves by a single symptom. We might however imagine that this sclerosis of the posterior columns could determine the phenomena of motor ataxy in the upper extremities, but it is not so; and this is explained by the separation which the inter-

*As for the articular alterations, which are of frequent occurrence, they depend evidently only upon the immobility, and differ in no respect from those which are produced by that cause, outside of any influence of the nervous system. Upon this subject consult Teissier, Memoires sur les effets de l'immobilité longtemps prolongée des articulations. Lyons, 1844.

†According to M. Routier, there should be a dimunition of the temperature of the paralyzed side immediately after the attack. The elevation of the temperature only comes on at the end of twelve or twenty-four hours. (A. Routier, Théses. Paris, 1846.)

mediate posterior sulcus establishes between the centripetal fibres of the pelvic and those of the thoracic extremities. Even in cases of compression of the cord in the cervical region, when the secondary sclerosis also affects the external fasciculus of the posterior column, it is probable that we should not find symptoms of ataxy. In fact, ataxy supposes the destruction of a certain number of nerve tubes, and secondary sclerosis does not appear to destroy the healthy tubes which are plunged into its interior; it only deforms them and may exalt their activity, but does not annihilate them.

In conclusion, I will state that these cases which I have gathered at Sainte-Eugénie, with M. Triboulet, and two others which I have studied at the Salpétrière with M. Charcot, warrant me in affirming that a cure is possible, even when the columns of the cord seem to have undergone secondary degeneration. In these five cases there was complete paraplegia due to the compression of the cord by Pott's disease. In four cases, sensibility and motion have returned in all their integrity; in only one, motion, without having recovered its entire liberty, nevertheless allows the patient to walk. In this case the paraplegia was flaccid; in the others it was accompanied with contraction. We may therefore conclude that the nerve tubes of the cord may be regenerated like those of the peripheral nerves, not only in the child but also in the adult, and even when the degenerated fasciculi have already been the seat of a hypergenesis of nuclear elements.

SECONDARY DEGENERATIONS OF THE SPINAL CORD.

EXPLANATION OF THE PLATE.

FIG. 1. Secondary degeneration of the mesocephale in an old softening of the right hemisphere. Atrophy and gray color of the right peduncle. Flattening of the Pons Varolii on the right side. Atrophy and gray color of the right anterior pyramid. Gray discoloration in the left lateral column below the bulb.

FIG. 2. Histological lesions in the first stage of secondary degenerations.

a. Granular bodies.

b. Vessel with fatty granulations accumulated in the lymphatic sheath.

c. The same granulations, but more numerous, in the lymphatic sheath at the point of bifurcation.

FIG. 3. Histological lesions in the later stages of secondary degenerations.

a. Myelocyte.

b. Vessel with numerous nuclei and very few fatty granulations in the sheath.

c. Amyloid body.

FIG. 4. Section of the cord in the dorsal region in a case of old secondary degeneration of the posterior columns.

ac and *a'c.* The posterior columns.

bc and *b'c.* The portion of these columns where the tubes are scanty, separated by the connective tissue of new formation.

FIG. 5. Sections of the cord in a case of old lesion of the left hemisphere. The parts colored black indicate the points of location of the secondary degeneration.

a. Cervical enlargement.

b. Dorsal region.

c. Lumbar enlargement.

FIG. 6. Descending degeneration in a case of compression of the cord at the upper part of the dorsal region.

a. Section made a few centimeters below the compression.

b. Inferior portion of the dorsal region.

c. Lumbar enlargement.

FIG. 7. Ascending degeneration in a case of compression of the cord at the lower part of the dorsal region.

a. Section made a few centimeters below the compression.

b. Superior portion of the dorsal region.

c. Middle of the cervical enlargement.

d. Superior portion of the cervical enlargement.

FIG. 8. Secondary degeneration in a case of compression of the cauda equina.

a. Inferior portion of the lumbar enlargement.

b. Superior portion of the lumbar enlargement.

c. Middle of the dorsal region.

d. Middle of cervical enlargement.

AGE.	DURATION.
66	4 m
69	3 yr
50	18 m
59	6 m
70	2 yr
44	5 yr
67	9 m
56	1 yr
67	3 yr
33	13 yr
70	sev.
42	14 m
56	18 yr
75	12 yr
66	5 yr
51	14 m
79	18 m
76	10 yr
40	1 yr
68	sev.
67	2 yr
69	1 yr
74	10 yr
66	3 yr
68	1 yr
77	sev.
72	2 yr
67	3 yr
61	3 yr
65	8 yr
76	2 yr
74	2 yr

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Fig. 1.

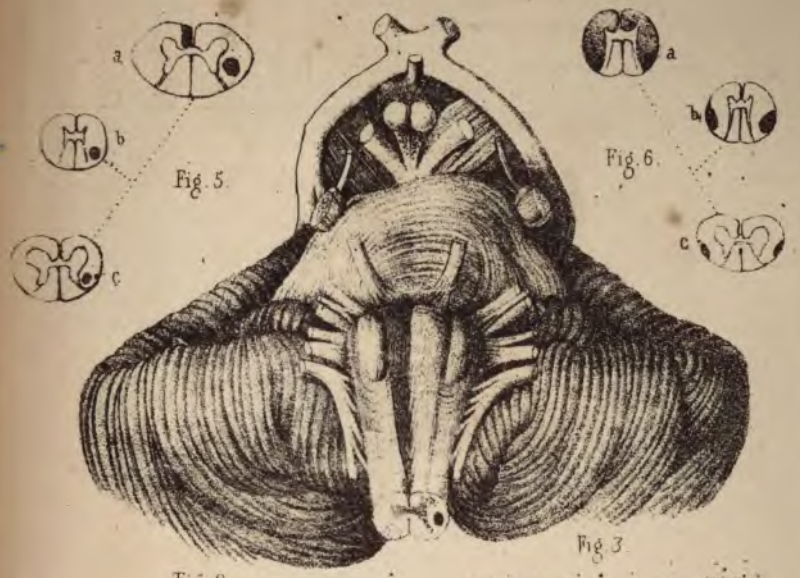


Fig. 5.

Fig. 6.

Fig. 3.

Fig. 2.

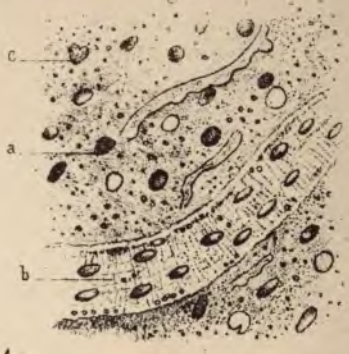
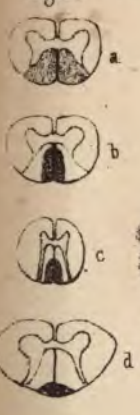


Fig. 4.

Fig. 8.



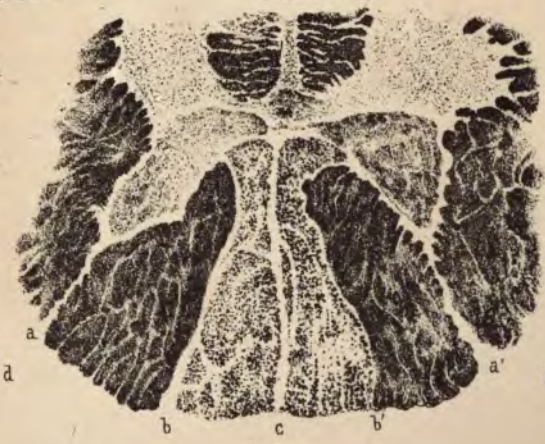


Fig. 7.

Léon Tripier del. Imp. Becquet, Paris. P. Lacherbauer